# A CLINICAL and PATHOLOGICAL REPORT

on

# 70 CASES OF STILL-BIRTH & NEO-NATAL DEATH,

with

# NOTES ON THE BIOCHEMICAL INVESTIGATION OF SOME OF THESE CASES

by

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# I N T R O D U C T I O N.

It is a common and universal experience that life is crippled or curtailed by the occurrence of disease, which leads to a greater or lesser degree of disablement, incapacity and premature death. The exigencies of the present time demand increasing industrial output and production, and in order to obtain this it is imperative that the care of the national health should be one of prime importance. The need for the application of methods of preventive medicine to infant mortality, tuberculosis, venereal diseases and to industrial welfare activities has therefore become more and more a matter of national concern.

The fall in the mortality of tuberculosis during the last fifty years is striking; from pulmonary tuberculosis alone the death rate fell from 2,653 per million of the population living in 1867 to 801 in 1924. This conquest is in a large part due to the preventative measures employed. Enumerated shortly these are:-

(1) Care of the tubercular patient by increasing his powers of resistance with suitable nutrition, open air, good housing, education and, where necessary, sanatorium treatment, or hospitalization.
(2) Care of the tubercular patient's family; this consists mainly in the routine medical examination of them at regular intervals, thereby attempting the possible detection of early stages of the disease.
(3) /

(3) Prevention of spread from the diseased to the healthy: and
(4) Control of the sale of tuberculous milk and meat.

The reduction in the still-birth rate, where adequate supervision is carried out, is equally striking. The figures for the Royal Maternity Hospital, Edinburgh for 1922, where the first Ante-natal Clinic was held, as quoted by Ballantyne[1] are

| | | |
|---|---|---|
| For the whole practice of the Royal Maternity Hospital, 156 still-births in 2710 births, | i.e. | 56.3 per 1,000 |
| For the intern department - - | | 68.8 per 1,000 |
| For the extern department - - | | 41.2 per 1,000 |
| For the venereal diseases department | | 157.8 per 1,000 |
| Receiving ante-natal supervision and treatment - | 50.7 per 1,000 | |
| Receiving no ante-natal supervision nor treatment - - - | 606.0 per 1,000 | |
| For the whole practice of the Hospital | | 56.3 per 1,000 |
| For 678 ante-natally supervised pregnancies (excluding venereal diseases) | | 5.9 per 1,000 |

The extremes 606 per 1,000 among expectant mothers suffering from venereal diseases, and with neither supervision nor anti-syphilitic treatment in their pregnancies and 5.9 per 1,000 among women free from venereal disease and supervised and, if necessary, treated in this time of expectancy, are interesting.

One further point of interest is that in spite of the reduction in the infant mortality rate as a whole during the last 20 years, there has been no reduction in this rate during the first month of life.

Pinard /

Pinard[2] pointed out many years ago that the preservation of child life must be considered in relation to three periods. (1) The pre-conceptional period, in which the seed to be sown and the soil in which it is to germinate are the elements concerned. (2) The intra-uterine period, in which development is carried from the blastocyte stage up to the fully developed infant. (3) The post-natal child. The second stage is the one which mainly concerns us.

The recent advances in midwifery shew that the care of the health of the pregnant woman is of primary importance to the production of the healthy child. During pregnancy the function of practically every organ in the mother's body is modified to meet new demands made upon it, and the extra strain thus thrown upon the system will soon break the weakest link in the chain. From the physiological point of view the pregnant woman shews certain variations, which require consideration. There is the retention of the products of metabolism for the building up of foetal and new maternal tissue. De Wesselow[3] believes that there is some evidence of alteration in the function of kidneys and liver, and if this be true, we have a connection link between normal and toxaemic pregnancies. The biochemical findings in pregnancy have recently been investigated. Urea and non-protein nitrogen /

nitrogen estimations of the blood and urine and urea concentration tests are of particular interest to us on account of the high still-birth rate in pregnancies complicated with albuminuria. Further urea estimations of the blood and urine have taken on a new significance since the probable relationship between nephritis and placental infarction, retro-placental haematoma and accidental haemorrhage was experimentally shewn by F.J. Browne[4] .

The following report is based on the findings in 70 cases occurring in the in-door and out-door practice of the Royal Maternity Hospital, Edinburgh, with the exception of two cases occurring in the in-door practice of University College Hospital, London. I am deeply indebted to the physicians of the Hospital for permission to do this work, and also for the facilities granted for the work being carried out. I am also indebted to Professor F. J. Browne for much helpful advice.

The method of investigation followed :

I. With reference to the foetus:
   (1) Postmortem examination of the foetus and the placenta: this included weighing of the foetal organs.
   (2) Microscopical examination of lungs (bases of lower lobes), umbilical cord and placenta in all cases; and of the liver, spleen, kidney, suprarenal, thymus and thyroid in all cases of syphilis or suspected syphilis.
   (3) Levaditi examination of foetal organs of cases of syphilis or suspected syphilis and of macerated foetuses.

II. /

II. With reference to the mother :
(1) Age; whether married or single.
(2) History of previous health, with special reference to the occurrence of scarlet fever, diphtheria, kidney disease, syphilis and gonorrhoea.
(3) Clinical examination of the mother - special attention was paid to the state of the blood pressure and to the condition of the teeth and gums.
(4) Wassermann reaction was taken.
(5) Blood urea nitrogen and non-protein nitrogen estimations and urea concentration tests were carried out on all in-patients, and where possible, on out-patients, where kidney lesion was suspected.
(6) Past obstetrical history, if any, was recorded.
(7) History of the present pregnancy and labour was obtained

The term "still-birth" is used in its widest sense to include infants born dead at and after twenty-eight weeks of pregnancy, as well as those born with the heart beating, but who failed to breath after complete birth of the head and body.

The term "neo-natal death" is used to include all infants in whom respiration took place after complete birth, even though the child only gasped a few times.

The work has been divided into sections,

I. HISTORICAL.
II. MACERATION OR ANTE-NATAL DEATH.
III. ASPHYXIA.
IV. TENTORIAL TEARS AND CEREBRAL HAEMORRHAGE.
V. PNEUMONIA.
VI. SYPHILIS.
VII. PREMATURITY.
VIII. FOETAL STATES.
IX. PROPHYLAXIS.
X. SUMMARY.
APPENDIX - CASE RECORDS.

References :

1. Ballantyne, J.W., Brit. Med. Jour., 1925, II. 585.

2. Pinard quoted by T. W. Eden, "Ante-Natal Care" Lancet, 1925, II. 1152.

3. De Wesselow and Wyatt, Toxaemias of Pregnancy.

4. Browne, F. J., "Etiology of Accidental Haemorrhage and Placental Infarction", Brit. Med. Jour., 1926, I. April, 17th.

# I. HISTORICAL.

"Yet I doubt not through the ages one increasing
" purpose runs,
"And the thoughts of men are widened with the
progress of the sun. Tennyson.
~~Bridges~~.

In going far back into the history of beliefs on ante-natal deaths and still-births, a great deal of interesting work and research can be done, particularly regarding old superstitions in this country and pagan beliefs in other countries,as in Polynesia and Ancient Egypt.

Among the early records one finds pagan superstitions associated with the birth of monstrous babes; famine and psetilence as well as peace and plenty were attributed to follow the birth of these infants.

There are several examples in the Old Testament of the belief in the power of an enemy's curse upon pregnant women. One example is the curse on Ephraim "Give them a miscarrying womb and dry breasts"[1] Shakespeare gives us an example of this curse in "King Lear".[2]

"Hear, nature, hear; dear Goddess hear !
"Suspend thy purpose, if thou dids't intend
"To make this creature fruitful
"Into her womb convey sterility.
"Dry up in her the organs of increase
"And from her derogate body never spring
"A babe to honour her. If she must teem
"Create her child of spleen; that it may live
"And be a thwart disnatur'd torment to her
"Let /

"Let it stamp wrinkles in her brow of youth
"With cadent tears fret channels in her cheeks.
"Turn all her mothers pains and benefits
"To laughter and contempt; that she may feel
"How sharper than a serpent's tooth it is
"To have a thankless child ! "

There are also superstitions regarding the most favourable day of the month to be born[3]: the 5th day of the month was unkindly and terrible because on the 5th Emriyses assisted at the birth of Hoseus: the 10th day was favourable for a boy to be born; the 4th for a girl; the 9th day of the first month was a good day on which to beget or be born, both for a male and a female, it was never a wholly evil day. This superstition[4] still survives to some extent among the fisher folk in Fifeshire. They believe that the tide is supposed to exercise an effect on labour: they believe that a child is born more easily during full-moon: children born at full-moon are also held to be lucky, while those born at the change of the moon are sickly and small.

The women who attended the Ante-natal clinic had many superstitions. One was that if a pregnant woman sat with her legs crossed she would cause a cross-birth. On looking through the literature it is interesting to find that the Romans were aware of this "truth". Pliny[5] mentions it, and the instance is that of Alcemena, who travailled with Hercules for seven days and nights because the /

the goddess Lucina, sat in front of the house with clasped hands and crossed legs.

Another superstition which survives from ancient times is associated with the cure of sterility and the prevention of obstetrical complications. In ancient Greece sterility was cured by bathing in the River Etatos in Arcadia; in Scotland we find records of St. Fillan's at Comrie and St. Mary's at Whitekirk, being used for the same purpose.

Passing from superstitions to actual observations, one finds that Aristotle gives accurate descriptions of monstrosities both animal and human, and displays a knowledge of the cause of the malformation[6]. Soranus - "the father of Obstetrics and Gynaecology" describes the determination of the sex of a child by the inspection of the mother's urine. The authors of the sixteenth and seventeenth centuries observed that in twin pregnancies there might be two placentae or only one[7]. Further, they observed, that when the foetuses were of the opposits sex they were always separated by a membrane, while of the same sex the membrane was sometimes lacking. Viandel naively regarded the difference as a divine interest safeguarding the morals of twins in utero.

From the beginning of the 18th century with the passing of the monopoly of obstetric practice from the hands /

hands of ignorant midwives to the care of trained obstetricians, there appeared a number of scientific works on monstrosities. In 1702 there appeared the first separate work on foetal disease as apart from those on monstrosities, the treatise of Düttel entitled "De morbus foetuum in utero materno" and presented for the degree in medicine at the University of Halle[8].

In 1828 Orphi[9] gives us an accurate description of the normal pathological appearances of the organs of the new-born babe. In 1858 King of London published the first paper in English on "The cause of death of the Foetus". He pointed out that still-births were more frequent in breech than in vertex deliveries.

By 1888 there had been great advances made in midwifery practice, and that interest had been awakened to the preventable aspect of a certain number of still-births is shewn by the choice of the subject for discussion at the Obstetrical Section of the British Medical Association in that year, namely, "Intra-uterine death, "its pathology and preventive treatment".

Recent authorities are too numerous to quote, but J. W. Ballantyne requires special mention as it is largely owing to his persistent advocacy and leadership that the present interest in ante-natal work and the causation of pre-natal, intra-natal and neo-natal deaths, has received /

received wide recognition. The close relationship between ante-natal work and deadbirth is shewn in the recent report of the Medical Research Council[10], where fully one half of the total deaths out of 1,673 cases of deadbirths and neo-natal deaths, were due to the various complications of labour and the toxaemias of pregnancy.

References :

1. Hosea IX. 14.

2. King Lear, Act I. Scene IV.

3. Garrison, F.H., Histroy of Medicine.

4. Rosie, D., Caledonian Medical Journal, July 1926.

5. Rosie, D., loc. cit.

6. Ballantyne, J.W., The Foetus.

7. Williams, J. Whitridge. "Note on placentation in quadruplet and triplet pregnancy" Bulletin of John Hopkins Hospital, Novr. 1926.

8. Quoted by J. W. Ballantyne, loc. cit.

9. Quoted by C. J. Thomson "The Causation of still-birth and neo-natal death" Jour. Obst. & Gynec. Brit. Emp. 1926, XXXIII, 3.

10. Child Life Investigations. Special Report Series No. 109.

TABLE I.

Macerated Foetuses.

| No. | Record No. | Age | Parity. | Duration of Pregnancy. | Sex & Weight. | A. N. care. | Signs and history of Syphilis. | Remarks. | Cause of Foetal death. |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 1 | 36 | 9 | 8 months | F. 2,350 | Yes. | None. Wassermann negative | | General foetal oedema. |
| 2 | 6 | 37 | 0 | 8 months | M. 1,770 | None | None. Wassermann negative. | Accidental haemorrhage. | Placental infarction and retroplacental haematoma. |
| 3 | 7 | 25 | 0 | 9 months | M. 2,800 | Yes (Irregular) | None. Wassermann negative. | | Placenta praevia. |
| 4 | 11 | 37 | 4 | 7½ mths. | M. 1,320 | Yes | None. Wassermann negative. | Trace of albumen on 3 occasions during pregnancy. B.P. not increased. Had low urea conc. test. | Placental infarction and fibrosis. |
| 5 | 12 | 27 | 1 | 9 months | F. 3,000 | None | No history. Wassermann reaction strongly positive in puerperium. Child showed evidence of syphilis. | | Syphilis. |
| 6 | 18 | 27 | 0 | 8 months | M. 2,130 | None | None. Wassermann negative. | Had 1.4% albumen in urine and high B.P. | Placental infarction and insufficiency. |

| o. | Record No. | Age | Parity. | Duration of Pregnancy. | Sex & Weight. | A. N. care. | Signs and history of Syphilis. | Remarks. | Cause of foetal death. |
|---|---|---|---|---|---|---|---|---|---|
| 7 | 20 | 27 | 0 | 8½ mths. | F. 2,280 | None | None. Wassermann negative. | | Lateral placenta praevia. |
| 8 | 31 | 27 | 1 | 7 months | F. 875 | None | None. Wassermann negative. | Twin pregnancy. Had albuminuria and high blood pressure. | Placental infarction |
| 9 | 38 | 37 | 4 | 8 months | M. 2,240 | None | None. Wassermann negative. | Concealed & accidental haemorrhage & lateral placenta praevia. Caesarean section performed. | Placental infarction |
| 0 | 41 | 27 | 4 | 8 months | F. 2,540 | None | Wassermann negative. | Concealed & revealed accidental haemorrhage. 0.2% albumen in catheter specimen. Caesarean section performed. | Placental separation and infarction. |
| 1 | 42 | 38 | 9 | 8 months | M. 2,650 | None. | Wassermann negative. | 0.2% albumen in catheter specimen. B.P. 120/86. | Albuminuria + retro-placental haematoma. |
| 2 | 43. | 27 | 0 | 7½ mths. | F. 1,030 | None. | Wassermann negative. | Had mitral stenosis and acute cardiac failure 3 wks. before delivery. | Asphyxia following failure in maternal cirulation. |

| o. | Re-cord No. | Age | Par-ity. | Duration of pregnancy. | Sex & Weight. | A. N. care. | Signs and history of Syphilis. | Remarks. | Cause of foetal death. |
|---|---|---|---|---|---|---|---|---|---|
| 3 | 45 | 20 | 0 | 7 months | F. 910 | None. | Wassermann negative. | Twin pregnancy with hydramnios No albumen, B.P. 130/80. | Placenta infarction. |
| 4 | 49 | 34 | 2 | 8 months | M. 1,610 | None. | Wassermann negative. | Accidental haemorrage and albuminuria with high blood pressure. | Placental infarction |
| 5 | 51 | 28 | 0 | 7½ mths. | M. 1,100 | Yes | Wassermann negative. | Twin pregnancy. No albuminuria. | Placental insufficiency |
| 6 | 52 | 28 | 0 | 7½ mths. | M. 1,200 | Yes. | | Twin to last case. | Placental insufficiency |
| 7 | 56 | 41 | 7 | 9 months | M. 3,640 | None. | Wassermann negative. No sign of syphilis found in foetus. | No albuminuria. | Not ascertained. |
| 8 | 57 | 33 | 6 | 7 months | M. 810 | Yes. N.A.B. treatment. | Had syphilis and had intensive course of treatment. Developed acute yellow atrophy and died 2 hrs. after birth of foetus. | | Syphilis |
| 9 | 59 | 30 | 1 | 9 months | F. 4,010 | None. | Wassermann negative. | No albuminuria. | Not ascertained. |

| o. | Re-cord No. | Age | Par-ity. | Duration of Pregnancy. | Sex & Weight. | A. N. care. | Signs and history of Syphilis. | Remarks. | Cause of foetal death. |
|---|---|---|---|---|---|---|---|---|---|
| 0 | 61 | 36 | 7 | 7½ mths. | F. 1,710 | None. | Puerperal Wassermann reaction +++. Foetus spirochaete +. | | Syphilis. |
| 21 | 65 | 28 | 2 | 7½ mths. | M. 1,230 | Yes. | Wassermann negative. | High blood pressure and albuminuria. | Placental infarc-tion. |
| 22 | 70 | 35 | 3 | 7 months | M. 870 | Yes. N.A.B. | Wassermann strongly positive. History of specific infection. | | Syphilis. |

## II. MACERATION or ANTE-NATAL DEATH.

Etiology and pathogenesis :

An analysis of table 1 shews that the cause of maceration in the 22 cases was :

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 1. Placental infarction | 11, | 18, | 31, | 38, | 41, 45, 49, 65, | 8 | cases |
| 2. Retroplacental haematoma | 6, | 42 | - | - | - | 2 | " |
| 3. Syphilis | 12, | 51, | 52, | 57, | 61, 70 | 6 | " |
| 4. Placenta praevia | 7. | 20 | - | - | - | 2 | " |
| 5. General foetal oedema | 1 | - | - | - | - | 1 | " |
| 6. Maternal cardiac condition | 43 | - | - | - | - | 1 | " |
| 7. Not ascertained | 56, | 59 | - | - | - | 2 | " |
| | | | | | | 22 | " |

1. Placental infarction :

The single cause, which is responsible for the largest number of cases in this group is placental infarction; but such a statement is by no means a dismissal of the matter, because, bound up with it are the factors which are responsible for the production of placental infarction. These factors are, unfortunately, difficult in their investigation and elucidation. The points requiring consideration are (1) the occurrence of physiological infarction, and (2) the maternal clinical findings and past obstetrical history in the cases where pathological infarction occurs.

A certain amount of infarction is physiological. The placenta is "an organ which after serving its purpose, "is cast off by the organism. Degeneration is the necess- "ary preliminary of shedding, and merely indicates a "progressive /

"progressive diminution of vitality towards the close of "its period of existence".[1] During the last two months of pregnancy these degenerative changes are found in the placenta; they consist in a gradual development of end-arteritis obliterans in the stem arteries with consequent diminution of blood supply of the villi, resulting in atrophy and degeneration; numerous small, firm, white patches are thus formed, usually in the spongy layer. A coagulation necrosis and fibrin network is frequently seen on the uterine surface of the mature placenta, either alone or in combination with these white patches.

Pathological infarction is an exaggeration of physiological infarction; it usually also occurs earlier in the pregnancy than physiological infarction; further in considering whether the condition present is pathological, the factor of foetal maceration must also be taken into account. T.W.Eden[2] believes that infarction undoubtedly increases in extent after the death of the foetus.

There are two types of infarction seen in pathological infarction (1) red infarction, and (2) white infarction. Some authorities hold that red infarction is the first stage of white infarction. Young and Miller[3] say "in many instances the local impairment "of the blood supply is due to thrombosis. This "thrombosis /

"bosis leads to (1) coagulation in the intervillous "spaces, (2) congestion of the vessels in the foetal "villi, and (3) necrosis". These stages give the characteristic naked eye appearances of red and white infarctions. The association of thrombi and infarcted areas in placentae was noted by F. J. Browne[4] in his experimental investigation of the etiology of accidental haemorrhage and placental infarction.

The frequent absence of placental infarction in syphilitic placentae where enderteritis obliterans is well marked, throws doubt on the relationship between endarteritis obliterans and placental infarction.

The association of placental infarction with other abnormal conditions of the placenta, viz., intra-placental and retro-placental haemorrhage, has been noted clinically and experimentally. In this group five out of the ten cases had haemorrhage and infarction, three of these being severe cases of accidental haemorrhage.

Winter[5] in 1885 first directed attention to the frequent association of albuminuria and accidental haemorrhage. Gordon Ley[6] from an examination of fifty cases ascribed the cause of accidental haemorrhage to a toxaemia of pregnancy, which damages the liver and the liver and the kidneys as well as the uterus. An ad-

vance /

vance in our knowledge of the etiology of placental infarction and accidental haemorrhage was made recently by their production experimentally. They were produced in 100% of pregnant rabbits investigated "by the setting "up of a chronic oxalate nephritis and then causing an "acute exacerbation with oxalates plus organisms. Fur- "ther in a certain percentage of cases placental infarc- "tion was produced by the introduction of organisms alone "after a chronic nephritis had been set up"[7].

In the present series of cases 7 out of the 8 cases had albuminuria. The exception (case 45) was a primigravida, aet 20, twin pregnancy at the 32 - 34th week. In this case there was no retention of urea in the blood, the urea concentration test was normal, the blood pressure was not raised, and the Wassermann reaction was negative. This was the youngest patient in the series; the ages of the others were, three at 27, one at 32, one at 31, and two at 37 years. The absence of signs of toxaemia in the average case of albuminuria of pregnancy, both clinically and biochemically, is impressed upon everyone who works in ante-natal wards. The exception to this general statement is oedema, which is found in a very large majority of the cases. In this group, however, cases 18 and 31, had oedema and toxaemic symptoms. In both oedema was present for three weeks before /

before they went into premature labour, the one at the 36th week and the other at the 31st week of pregnancy; both had high blood pressures, a considerable amount of albumen in their urine (1.2 and 0.8 % respectively), and pre-eclamptic symptoms, i.e., headaches and visual disturbance. Case 18 was a primigravida and case 31 was a one para whose previous pregnancy had ended prematurely. There was no history of toxaemic symptoms or signs associated with that pregnancy. Neither had urea retention in the blood and in both cases the urea concentration test result was between 2 and 3 mg %. In short both these cases had all the features which one associates both clinically and biochemically with albuminuria of pregnancy.

Case 41 had 0.2% albumen in the urine on admission and a severe concealed and revealed accidental haemorrhage - she was too ill for investigation on admission and died in hospital before it was considered advisable to carry the tests out.

The remaining cases - 11, 38, 49, and 65 - were multiparae. None gave a history of toxaemia in previous pregnancies. Case 11 had three previous full-time pregnancies and one abortion, which she attributed to carrying heavy weights while doing war work. She was under observation during the last four months of her pregnancy and /

and a trace of albumen in a catheter specimen of urine was noted on several occasions. She had treatment for a Bartholinian abscess and cystitis. Case 38 had three previous full time pregnancies and one miscarriage. Cases 49 and 65 had two previous full-time pregnancies. Cases 38 and 49 had accidental haemorrhage, revealed and concealed, associated with the placental infarction, and as the first sign of toxaemia: both had a considerable quantity of albumen in the urine, viz., 1.2 and 0.8 %. Case 11 went into premature labour about the 32nd week of pregnancy, three weeks after she had felt foetal movements cease: cases 38 and 49 went into labour about the 38th week immediately after accidental haemorrhage occurred. The feature which cases 11,38 and 49 had in common was a low urea concentration, between 0.7 and 1.75 mg %, ~~which~~ this is usually associated with a chronic nephritis. Case 11 had three of the features associated with chronic nephritis and pregnancy, i.e., a small quantity of albumen in the urine on several occasions, persistently low blood pressure, and low urea concentration. Cases 38 and 49 had only low urea concentration. A low blood urea was found in all the cases. One would be inclined to classify case 11 as a chronic nephritic, but cases 38 and 49 require further observation before a definite conclusion can be arrived at. In case 65 no urea concentration was carried out as the patient was delivered /

delivered on the "district".

The finding arrived at from the investigation of these 8 cases of placental infarction is that in 7, viz., 11, 18, 31, 38, 49 and 65, there was evidence of kidney damage. The extent of the kidney lesion was not sufficient to cause a urea retention in the blood, but the extent of the placental lesion was sufficient to cause the death of the foetus. 2 of the 5 cases in which the urea concentration test was carried out had pre-eclamptic symptoms, but normal tests, while the remaining three cases had low urea concentrations. Two of these three cases had severe accidental haemorrhage. The value of these tests will be considered in the section on Prophylaxis, as one considers that the number of cases in each group is too small to justify any conclusions being drawn.

2. Retro-placental haematoma :

There are two cases due to this cause. Case 6 was a primigravida aet 37, She was admitted to hospital four days before going into premature labour on account of antepartum haemorrhage; on the second day after admission there was a trace of albumen in the urine, and the blood pressure was $^{130}/_{90}$ mm.Hg. No albumen was found in the patients urine again during her stay in hospital. There was no retention of urea in the blood, and /

and the urea concentration test, carried out on the tenth day after delivery, was normal viz., between 2 and 3 mg %. Case 42 was a 9 para, aet 38, and was admitted to hospital with antepartum haemorrhage: there was 0.2% albumen in the urine and the blood pressure was $^{126}/_{90}$ mm.Hg. Albumen was present in the urine during the whole of her stay in hospital, but no investigations were carried out. Case 6 was associated with placental infarction; Case 42 was not associated with placental infarction. Both cases had intermittent haemorrhage: Case 6 went into labour on the 4th day after the first haemorrhage, and case 42 within 24 hours of the first haemorrhage.

The post-mortem findings in these two cases were :- slight degree of maceration, labour having commenced shortly after the retro-placental haemorrhage had occurred. The skin was wrinkled and sodden, and pink in colour, in place of the deep purple which one finds in fresh foetuses. There was little vernix caseosa. The tissues were infiltrated with blood-stained serum, and there were numerous petechial intra-thoracic haemorrhages. The intra-thoracic haemorrhages were more marked in these two cases than in any of the other cases seen. Sub-pleural haemorrhages were found along the margins of the lobes of the lungs, most marked in the upper /

upper lobes posteriorly, and around the hilus of the lung; sub-epicardial haemorrhages were most marked around the interventricular sulcus anteriorly and along the great vessels; subcapsular haemorrhages occurred most frequently on the posterior surface of the thymus gland, and there were also a few haemorrhages along the posterior thoracic wall and on the upper surface of the diaphragm. The large number of inter-thoracic haemorrhages is due to the acute asphyxiation of the foetus when the foetal blood supply was cut off by the retro-placental haematoma.

3. Syphilis :

Syphilis is the cause which was responsible for the second largest number of ante-natal deaths. The total number of cases is 6, or 8.5%. There were only two cases of certain syphilis, i.e., spirochaete positive. The fact that the Levaditi method only, and not the dark ground method, was used, may to a certain extent account for the low percentage of cases in which the spirochaete was found.

Maceration was advanced in 4 of the 6 cases. Numerous observers have noted that the degree of maceration met with in syphilitic foetuses is frequently more advanced than that found in foetuses, where ante-natal death was due to e.g., infarction, and have attributed /

attributed this to the absence of stimulus to the uterus to contract.[8] The maceration renders histological investigation of the foetal organs useless, and the diagnosis rests on :

(a) Examination of the foetal organs and of the umbilical cord for spirochaetes.

(b) Histological examination of the placenta, cord and epiphyses.

(c) Weights and weight ratios of the placenta and of the foetal organs.

(d) Maternal Wassermann reaction;

(e) History or signs of infection in the parents: and

(f) Past obstetrical history of the mother.

(a) Spirochaetes :

Spirochaetes were found in the foetal organs of cases 12 and 57. An exhaustive search was made of the umbilical cord in every case close to the foetal end, but no spirochaetes were found. The examination of the placenta for spirochaetes was also negative. The great difficulty in finding spirochaetes in some foetuses which shew the so-called "secondary signs" of syphilis, has been noted by numerous observers; further it has been noted that when the spirochaetes are present in macerated foetuses they generally occur in large numbers. The first finding is held to be due to the fact that the spirochaete pallida exists at times in /

in the form of chromidian granules, and the second finding is believed to be due to the spirochaete multiplying under anaerobic conditions in the dead tissues.[9]

(b) Placenta.

The placenta was obtained in every case for examination, and four out of the six shewed typically syphilitic changes;- uniform enlargement of the villi, most marked in the terminal villi, due to the proliferation of the subendothelial connective tissue in the villi, diminished intervillous spaces, and in parts, diminished blood supply. In some cases endarteritis obliterans affecting the main arteries, was a prominent feature, but in other cases it was no more marked than in an ordinary non-syphilitic placenta. Hyaline degeneration of the chorion was present in a few cases, but it was found present to an equal extent in non-syphilitic placentae. In none of the cases was a node of leucocytes found either in the chorion or in the decidua. Case 57 shewed irregular enlargement of the terminal villi, no diminished blood supply, and marked leucocytic infiltration of the chorion. Case 61 shewed no enlargement of the villi in the sections examined.

Umbilical Cord :

Cases 12, 51 and 52 shewed lymphocytic infiltration of /

of the media of the veins and arteries, and some proliferation of the connective tissue of the media and intima. Case 57 had slight lymphocytic infiltration of the media of the vein only. Cases 61 and 70 shewed no lymphocytic infiltration but partial occlusion of the lumen and endarteritis of the vessel walls. This condition was also found in non-syphilitic cases where the maceration was of some duration.

Chondro-epiphysitis :

This was diagnosed in only one case, viz., case 12, on naked eye examination, but was revealed on microscopic examination in every case. Case 52 was the only doubtful epiphysitis as deepening and irregularity in the size of the cells of the zone of provisional calcification alone was present.

(c) Weight ratios :

The weight ratios of the foetal organs is discussed fully in the section on syphilis. Those of the liver, spleen and placenta, may assist in the diagnosis of syphilis in macerated spirochaete-negative cases. In only one case, 12, of this series were they of value, the ratios in this case being liver 14.6, spleen 156, and placenta 4.1. The other cases recorded were all under eight months maturity, and cases 51 and 52 and 70 had probably been dead for a considerable time in utero. /

utero, and the organs had undergone considerable autolytic changes.

(d) Wassermann re-action :

The result of the Wassermann test is generally held not to be reliable in pregnancy. A triple positive re-action is said to occur more frequently in pregnant than in non-pregnant women, because the serum of the pregnant woman possesses an "irritability" which renders it more susceptible than ordinary serum to the variable conditions of handling.[10] As a result of this irritability anti-complimentary sera is said to occur eight times as frequently in pregnant as in non-pregnant women. There are various theories advanced to explain why a syphilitic pregnant woman has occasionally a negative Wassermann re-action. The most generally quoted one is Routh's;- namely, that the chorionic ferments prevent the spirochaetes from developing beyond the granule stage, and that even if the latter are allowed to pass the placental barrier into the maternal tissues, they are maintained in the granular phase in which they are unable to give rise to the tissue destruction which results in the liberation of proteins, the presence of which in the serum is the probable basis of the Wassermann re-action.

F. J. Browne[11] investigated the Wassermann re-action /

reaction in 100 cases of syphilis in pregnant women, and concluded that pregnancy has little or no influence in modifying the Wassermann re-action.

Boas, Gammeltoft and Sieck[12] have more recently investigated and followed up their cases two years after the original test in pregnancy, and found that the Wassermann re-action was as dependable in pregnancy as at other times.

I investigated the results of the Wassermann re-action obtained from 2,000 consecutive pregnant women who attended the outpatient clinic at the Royal Maternity Hospital, and found a triple positive re-action in 3.6% of the cases; a moderately strong re-action in 0.3%; a weak positive re-action in 1.4% and a negative re-action, with clinical evidence of syphilis, in 0.7%. Of these 120 cases, 35 had been under observation since their pregnancy at other venereal diseases treatment centres, and the diagnosis of syphilis was confirmed. 17 of the cases in the triple positive group had repeated strong Wassermann re-action as the only sign of syphilis, 6 of these 17 are included in the 35 cases who had been under observation for three months. 2 cases with a triple positive Wassermann re-action on one occasion only, were found to be non-syphilitic. These two cases helped to remind us that the Wassermann re-action /

re-action is not infallible, and also of the rule suggested in the interim report of the Medical Research Council examining the question of the value of the positive Wassermann reaction, that when a positive Wassermann re-action is the sole evidence of syphilis, detected by a particular observer, who makes the clinical examination, it should be repeated, and if still positive, it may then be accepted as proof of syphilis.[13]. From these results one concluded that the Wassermann re-action was of value in pregnancy, and that by its routine use syphilitic cases, which shew no other signs, can be detected.

Four out of the five mothers in this series of macerated foetuses had a positive Wassermann re-action, The exception was the mother of cases 51 and 52, whose Wassermann re-action was negative both before and after delivery. She shewed none of the usual features of congenital syphilis, but she was deaf, and her mentality was distinctly sub-normal. The histological examination of the placenta, cord and epiphysis were the most definite seen in the series.

(e) History or signs of infection :

This will only be referred to briefly here, as it is discussed at more length in the section on Syphilis. A history of infection was obtained in only one case (70) In this case there had been a primary sore, followed by a generalized rash and sore throat nearly four years before /

before her attendance at the clinic. No signs other than scarring from the primary sore were found on examination. The clinical histories and investigation of the remaining four mothers were negative. The Wassermann re-action was strongly positive in every case, except in the mother of cases 51 and 52. This mother was a primigravida aet 28.

(f) Obstetric history :

The relationship between the date of infection and the birth of premature and mature infants, still-births or ante-natal deaths, is generally believed to be definite; namely, the more recent the date of infection the more virulent the organism, and the more likelihood there is of premature termination of the pregnancy and conversely, the more distant the date of infection the more likelihood there is of the foetus being carried to term or of the occurence of a "latent" interval, when a healthy non-syphilitic child may be born. Case 70, the only one in this group where a history of infection was obtained, is a typical example of the occurrence of premature interruption of the pregnancy after a recent infection, followed in the subsequent pregnancy by a prolongation of gestation and ante-natal death. The history of case 61 is interesting: the first four pregnancies were normal full-time, children now alive and /

and well. The fifth pregnancy terminated at the 8th month, child lived 24 hours. The sixth pregnancy terminated at the fourth month. The seventh went to term, child now alive and well. During the seventh pregnancy the mother attended the ante-natal clinic where her Wassermann re-action was found to be weak positive before delivery, but negative after delivery; further, on looking up the case record, one found that the placenta weight ration was 2, there was no note of placental infarction or haemorrhage, and no history of toxaemia. The present pregnancy, the 8th, occurred one year after the 7th; there was no ante-natal supervision, and the mother was delivered on the "district" of a macerated spirochaete-positive foetus, and the maternal Wassermann re-action was strongly positive. It is not known when the infection occurred in this case, but if the patient was infected between the fourth and fifth pregnancies, than the premature interruption of the fifth and sixth pregnancies may have been due to this cause, and the seventh would then be an example of the occurrence of a latent interval. The infection may not, however, have occurred until after the 7th pregnancy, but the weak positive Wassermann re-action and the placental weight ratio in that pregnancy, may have some significance. Nothing is gained by speculation in these cases, but that /

that exceptions to the general occurrence of premature termination of a pregnancy after a recent infection I was able to observe in the series of cases with positive Wassermann re-actions, which were examined. Mrs H - came to the clinic with a history of two previous full-time pregnancies, children now alive and well. Her Wassermann re-action was tested as a routine procedure, and found to be moderately strong positive, On questioning a history of primary sore on the vulva (scarring of which was seen on examination) during the early part of the first pregnancy, was obtained, and the sore was followed by a generalized rash and sore throat. The sore disappeared with local application, but no anti-syphilitic treatment was received. The Wassermann re-action was repeated after a provocative dose of novarsenobillon and found strongly positive on two occasions. This case shews that one cannot rely on a bad obstetric history in all cases.

The mother of cases 51 and 52 was a primigravida, and the mother of case 12 was a 1-para - her previous pregnancy had terminated at the 8th month, $2\frac{1}{2}$ years previously, child now alive and well. An obstetric history was not obtained in case 57, as the woman was comatose on admission and died shortly afterwards. Her friends knew that the previous pregnancy had terminated in a still-birth.

4. Placenta Praevia :

Two cases were due to this cause following asphyxia from placental separation. The maceration was not advanced in either case, labour having commenced shortly after the fatal haemorrhage occurred. Case 7 shewed numerous intra-thoracic haemorrhages and a sub-capsular haemorrhage in the right suprarenal gland. In case 20 the maceration was more advanced, no intra-thoracic haemorrhages were present, but there was a haemorrhage into the right suprarenal gland.

5. Maternal cardiac condition.

Case 42 was due to this condition. The mother had severe mitral incompetence with acute failure of compensation three weeks before delivery.

6. General Foetal oedema :

This condition was responsible for one case.

7. Not ascertained :

In cases 56 and 59 the cause of ante-natal death was not determined. Both these foetuses were at full-term. Case 56 weighed 3,690 grammes, length 52.5 cms; and case 59 weighed 4,010 grammes, length 50 cms. The Wassermann re-action was negative in both cases, and the blood pressures were normal. None of the secondary signs of syphilis were found in either of the foetuses. The obstetric history of the mother of /

of case 56 was good, namely, six previous full time pregnancies, children all alive and well. Case 59 had one previous normal pregnancy, child now alive and well.

Symptoms and signs of ante-natal death :

The histories of these cases are given fully in the case records. A short summary of the findings shews that four out of six primigravidae gave a history of cessation of foetal movements, as the only indication of foetal death. One out of three 1-para stated that the foetal movements were not so active during the second last week, and that no foetal movements were felt in the last week of pregnancy. Neither of the 2-paras gave a history of cessation of foetal movements. The 3-para came to the clinic because she had not felt life for some time, and because she was getting smaller. One of the 4-paras noted that she had not felt foetal life for three weeks before delivery, and that she had felt a slow movement in the lower abdomen when she lay down at night. The two 7-paras definitely stated that they had not felt foetal life during the whole of the pregnancy. In three cases active foetal movements were felt just before a vaginal haemorrhage occurred.

Polak[14] states that acetonuria is a sign of foetal death, but this is frequently present in normal pregnancies.

TABLE II.

Asphyxia Neonatorum.

| No. of case. | Age. | Parity. | Presentation. | Nature of delivery. | Duration of pregnancy. | Cause of Asphyxia. | Sex and Weight. | Antenatal care. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| 2 | 34 | 0 | Breech | Breech hook for buttocks extraction (easy) of head | 9 months. | Breech | M. 3,620 | None | Small incomplete tear of right tentorium cerebelli. |
| 3 | 30 | 3 | Vertex podalic version done. | Breech | 8 months | Placenti praevies | M. 1,940 | None | |
| 4 | 39 | 0 | R.O.A. | Spontaneous. | 9 months | Not apparent | F. 3,680 | None | Haemorrhage in right suprarenal capsule. |
| 13 | 32 | 1 | Vertex | Caesarean section. | 7½ mths. | Placental infarction. | M. 1,440 | Practically none. | Albuminuria of pregnancy with concealed and accidental haemorrhage. |
| 19 | 38 | 0 | Vertex | Spontaneous. | 9 months | Not apparent. | F. 2,750 | None | Microscopic examination oflung revealed marked distension of alveoli with liquor amnii, lanugo hairs epidermal cells. |

| No. | No. of case. | Age. | Parity. | Presentation. | Nature of delivery. | Duration of pregnancy. | Cause of asphyxia. | Sex and weight. | Antenatal care. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 6 | 22 | 31 | 2 | R.A.A. Bipolar podalic version done. | Spontaneous of breech extraction of shoulders & head. | 9 months | Breech | M. 3,370 | None | Blood stained serum over base of brain - probably followed asphyxia. |
| 7 | 24 | 29 | 1 | Breech | Spontaneous of body. Extraction of shoulders. Craniotomy. | 9 months | Pressure of uterus and placenta on foetal head. | F. 3,520 gms. without brains. | None | Hydrocephalic foetus Wassermann negative. |
| 8 | 30 | District case. | | Vertex | Spontaneous. | 7 months | ? prolonged labour. | M. 1,085 | None | Haemorrhage into middle third of falx cerebri and massive interstitial haemorrhages in lungs. |
| 9 | 32 | 29 | 0 | Vertex | Spontaneous. | 9 months | Prolonged labour. | F. 2,855 | None | Incomplete tear of right tentorium cerebelli. |
| 10 | 33 | 22 | 0 | Vertex | Low forceps | 9 months | Prolonged 2nd stage. | M. 3,430 | None | Alveoli of lungs full of liquor amnii epidermal cells and lanugo hairs also polymorphonuclear infiltration in parts. |

| No. | No. of case. | Age. | Parity. | Presentation. | Nature of delivery. | Duration of pregnancy. | Cause of asphyxia. | Sex and weight. | Antenatal care. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 34 | 38 | 6 | Transverse Bipolar podalic version. Cord prolapsed. | Breech. Spontaneous. | 8 months | Prolapse of cord. | F. 1,350 | Since 4th month of pregnancy. | Had albuminuria (recurrent) |
| 2 | 46 | 38 | 5 | Vertex. Bipolar podalic version done. | Breech. | 8½ mths. | Placenti praevia | M. 2,445 | None | Only one artery in umbilical cord. |
| 3 | 53 | 28 | 3 | Vertex. | Spontaneous. | 9 months | Prolapse of cord. | M. 3,750 | None | Minor degree of hydronephrosis of both kidneys. |
| 4 | 58 | 22 | 2 | Vertex | Spontaneous. | 8 months | ? placental separation. | M. 2,400 | None | Horse-shoe shaped kidney. |
| 5 | 60 | 20 | 0 | Breech | Spontaneous of breech. Extraction of head. | 9 months | Breech | | | |
| 6 | 66 | 40 | 6 | Breech | Spontaneous. | 9 months | Breech | F. 3,350 | None | Twin pregnancy. |
| 7 | 68 | District case. | | Vertex born before arrival of nurse. | Spontaneous | 8½ mths. | Not ascertained. | F. 2,390 | None | |

## III. ASPHYXIA NEONATORUM.

Asphyxia was responsible for 17 or 24.2% of the total cases. As with the term "placental infarction", so with the term "asphyxia", the subject of causation is by no means dismissed by this simple statement. The cases are tabulated in table 2. From an examination of this table it will be seen that ante-partum asphyxia occurred in four cases, and intra-partum asphyxia in thirteen cases. The primary presentation in 12 cases was vertex, in three cases it was breech, and in 2 cases it was transverse. One case was delivered by low (easy) forceps, one by craniotomy, one by Caesarean section, and in 6 cases manipulations, either podalic version or extraction of a breech, were carried out. The remaining 8 cases were delivered spontaneously.

The most helpful information in the prevention of asphyxia neonatorum is obtained by investigating the cases according to the maturity of the foetus, taking the three groups, (1) Mature; (2) 8 - 8½ months; and (3) 7 - 7½ months.

(1) Mature foetuses : Cases 2, 4, 19, 22, 24, 32, 33, 53, 60, 66. There are 10 cases in this group, 5 were vertex presentations, 4 were breech presentations, and one was a transverse. Of the five vertex cases

4 /

4 occurred in primigravidae, whose ages were 39, 38, 29 and 22 years. Labour was not prolonged in cases 4 and 19, where the ages of the mothers were 39 and 38. In the first case the first stage of labour lasted 15 hours, and the second stage 1½ hours, while in the second case the first stage lasted 18 hours, and the second stage 4½ hours. The delivery was spontaneous in both cases: no sign of foetal distress had been noted before delivery. Neither of the foetuses were unduly large, their weights were 3,680 grammes, and 2,750 grammes. The post-mortem findings in case 4 were marked lividity, congestion of the internal organs, free straw-coloured fluid in the pleural and pericardial cavities, few subepicardial and a subcapsular haemorrhage in the right suprarenal gland. The suprarenal haemorrhage was not the cause of death, as this condition is associated with pallor of the mucous membranes and organs. The findings in case 19 were those associated with asphyxia - haemorrhages were numerous and extensive in the thorax. Microscopic examination of the lungs shewed liquor amnii, epidermal cells, meconium, and lanugo hairs. It is difficult to say why asphyxia occurred in these two cases. Asphyxia is known to occur more frequently in primagravidae than in multiparae, but there is usually some condition, such as prolonged labour, to account for it.

The /

The other two cases of vertex presentation in primigravidae were associated with prolonged labour. In case 32 labour commenced at 10 p.m. on 11:5:26; membranes ruptured at 11 p.m. on 12:5:26, and a still-born foetus was delivered spontaneously at 10.50 a.m. on 13:5:26. There was a small tear of the right tentorium cerebelli but this was not lethal. In case 33 the first stage of labour lasted 24 hours, and the second stage 7 hours. The membranes ruptured 20 hours before delivery. The foetus was delivered by low forceps on account of failing foetal heart. Neither of the foetuses were unduly large, their weights being 2,855 grammes and 3,430 grammes. There was no contraction of the pelves, therefore primiparity per se was probably the cause of the dystocia. Together with the prolonged labour in these cases the foetal head was subjected to an unusual amount of pressure on account of the rupture of the membranes 10 and 20 hours before delivery. In the vertex case which occurred in a multipara the cause of the asphyxia was prolapse of the umbilical cord. There was no pelvic contraction, nor any reason found to account for the prolapse.

Of the four breech presentations 2 occurred in primigravidae, 1 in a 1-para and 1 in a 6-para. The breech presentation in the 6-para was associated with a twin pregnancy, therefore, the possibility of premature separation /

separation of the placenta arises, and in this case it is justifiable to include the case as one due to an unavoidable cause. The breech presentation occurring in the 1-para was associated with hydrocephalous, delivery being effected by means of craniotomy. The patient was admitted to hospital with the foetus born as far as the umbilicus, and with umbilical cord pulseless. Prolonged pressure on the foetal head by the uterine wall and placenta had probably interfered with the foetal circulation and brought on the asphyxia. Secondary signs of syphilis were found in the foetus. The association between a large number of cases of hydrocephalous and syphilis has been pointed out elsewhere. The craniotomy had destroyed the brain tissue, and therefore one was unable to say whether the hydrocephalous was of the true congenital type or due to syphilis, - if of the former type, then the cause of asphyxia was unavoidable, but if of the latter type, it was avoidable. The remaining two breech cases occurred in primigravidae, neither of whom had any ante-natal supervision, and in both cases the foetal heart was not heard on admission to hospital. In case 2 the presentation was a frank breech, and considerable difficulty was met with in the extraction of the breech; the head was easily delivered. Case 60 was admitted to hospital with the foetus born to the umbilicus, and the umbilical cord pulseless. With ante-natal /

ante-natal supervision, these cases of still-birth would probably have been avoided, the malpresentation could have been diagnosed, and steps taken to rectify it. This statement applies equally to the remaining case in the group of mature foetuses, where the presentation was transverse, the patient being admitted to hospital with an arm prolapsed into the vagina.

(2) 8 - 8½ months maturity.

Vertex presentations - Cases 3, 46, 58, 68.
Transverse presentation Case 34.

All the mothers in this group were multiparae. There was a placenta praevia in cases 3 and 46, for which bi-polar podalic version was done. Case 46 had in addition only one artery and one vein in the umbilical cord, and this malformation may have played a part in the asphyxiation. King[15] reports two cases of intra-uterine death of the foetus at the 8th and 6th month of pregnancy where no other cause of death than this malformation was found. Case 58 was knocked down by a motor car and injured, and went into premature labour the same day. The birth of the placenta was preceded by two large blood clots; there was no clot passed after the birth of the placenta, which on naked eye and microscopic examination appeared normal. The probable cause of death in this instance was traumatic separation of the /

the placenta. The remaining case 68 occurred on the district, the foetus being born before the arrival of the nurses, and the labour was said to have been short. The cause of asphyxia was not ascertained.

The transverse presentation occurred in an albuminuria patient who had been under treatment by her own doctor since the 4th month of pregnancy. The cord prolapsed into the vagina when internal version was being performed, and was found to be pulseless.

(3) 7 - 7½ months maturity :

Vertex presentations, cases 13 & 20.

The cause of asphyxia in case 13 was accidental haemorrhage and placental infarction associated with albuminuria. The mother had been told by her doctor to take her urine for examination, but she had omitted to do so: the delivery was by Caesarean section. The cause of asphyxia was not found in the remaining case. It occurred on the district practice, and the haemorrages were the most extensive found in this series of cases. There was no evidence of syphilis.

A brief summary of these findings shews that 7, probably 8, of these cases might have been prevented if they had received ante-natal care. In 5 cases the cause was preventable according to our present knowledge, and in 5 cases the cause was not apparent. The outstanding features /

features of this group are (1) the lack of ante-natal care, and (2) the large number of mature foetuses. The diagnosis of the condition rests mainly on the foetal heart rate, and this may not, at times, have the significance it is supposed to have: Further, it is not always easy to listen to, and particularly to count the feotal heart rate when the patient is in the 2nd stage of labour and the uterine contractions are occurring frequently, and the patient is crying out. From this series of cases one thinks that the most hopeful means of prevention of asphyxia is closely related to the prevention of malpresentations in labour and in the prevention of too prolonged labour.

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TABLE III.

Tentorial Tears and Cerebral Haemorrhage.

| No. | Record No. | Age. | Parity. | Presentation. | Duration of Pregnancy. | Nature of Delivery. | Tear of Tentorium. | Cerebral haemorrhage. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 21 | 18 | 0 | Frank breech | 9 mths. | Bougie induction. Extraction. | Complete tear of left tentorium cerebelli Incomplete tear of right. | Diffuse haemorrhage over base of brain. | |
| 2 | 22 | 31 | 2 | R.A.A. | 9 mths. | Impacted shoulder. Bipolar podalic version. Extraction | None. | Diffuse blood-stained serum over base of brain. | Probably followed asphyxia. |
| 3 | 23 | | - | (Probably vertex) | 9 mths. | Spontaneous. | Incomplete tear of right tentorium cerebelli. | None | District case. No history obtained. |
| 4 | 32 | 29 | 0 | L.O.A. | 9 mths. | Spontaneous. | Incomplete tear of right tentorium cerebelli. | None | Asphyxia was probably the cause of death in this case. |
| 5 | 39 | 37 | 0 | Vertex | 9 mths. | Caesarean section. | Incomplete tear of cruciate ligament between 1st cervical vertebra. | Haemorrhage along anterior surface of spinal cord from level of 2nd -6th C. V. | |

| No. | Record No. | Age. | Parity. | Presentation. | Duration of Pregnancy. | Nature of Delivery | Tear of Tentorium | Cerebral haemorrhage. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| 6 | 44 | 17 | 0 | R.A.A. | 9 mths. | Impacted shoulder. Bipolar podalic version. Extraction. | Left tentorium cerebelli completely torn: incomplete tear of right tentorium cerebelli. | Blood clot over left tentorium Haemorrhage into middle third of false × cerebri. | |
| 7 | 46 | 38 | 5 | Vertex | 8½ mths. | Bipolar podalic version. Extraction. | None. | Blood stained serum in left temporo-sphenoidal region extending towards base of brain. | Asphyxia following placenta praevia. primary cause of death. |
| 8 | 47 | 20 | 0 | Vertex | 7½ mths | Spontaneous. Precipitate. | Tear of false cerebri. Complete tear of left tentorium cerebelli Bruising of right tentorium cerebelli. | Clot extending down false × cerebri, mainly on left side - extending down to tentorium and cerebellum on that side. | |

| No. | Record No. | Age. | Parity. | Presentation. | Duration of pregnancy. | Nature of Delivery | Tear of Tentorium | Cerebral haemorrhage. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| 9 | 55 | 25 | 0 | R.O.A. | 9 mths. | Low forceps (easy) | Complete tear of left: incomplete tear of right. | None. | |
| 0 | 58 | 22 | 2 | Vertex | 8 mths. | Spontaneous. | None. | Blood stained serum over base of brain. | Asphyxia ? cause of death. Horse-shoe shaped kidney. |
| 1 | 69 | 31 | 0 | Vertex | 9 mths. | Low forceps (easy) | Complete tear of right tentorium. | Clot overlying tear & cerebellum. | |

TABLE III. (B).

Cerebral Haemorrhage.

| Record No. | Age. | Parity. | Presentation | Duration of pregnancy. | Nature of Delivery. | Sex and Weight. | Antenatal care. | Cerebral haemorrhage. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| 5 | 24 | 0 | Vertex | 7½ - 8 months | Spontaneous. | F. 2,070 | None | Large blood clot in both lateral ventricles. | Child lived 4 hrs Massive interstitial haemorrhage in lungs - also sub-pleural, sub-epicardial, diaphragmatic etc. |
| 10 | 24 | 0 | Vertex | 8 mths. | Spontaneous | F. 1,440 | Yes. | Large clot 2½ x 3 cm. in right lateral ventricle: small clot in lat. left ventricle - haemorrhage into right olfactory tract optic chiasma, inferior surface ofright temporal lobe. | Syphilitic foetus |
| 14 | 30 | 2 | Breech | 7 mths | Spontaneous | F. 1,100 gms. | None | Small clot in Rt. lateral ventricle. Several subependymal haemorrhages in ventricles. | No haemorrhages seen on naked-eye examination but massive interstitial haemorrhage seen in lung on microscopic examination. |
| 25 | 19 | 0 | R.O.A. | 7½ mths. | Spontaneous. | M. 1,710 | None | Clot 1x 1½ cms. in right lateral ventricle, and several petechial subependymal haemorrhages. | Child lived 12 hours. Talipes equino varus of left foot. |

| | Record No. | Age. | Parity. | Presentation. | Duration of Pregnancy. | Nature of Delivery. | Sex and Weight. | Antenatal care. | Cerebral haemorrhage. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 5 | 27 | 39 | 0 | L.O.A. | 7 months | Spontaneous. | M. 700 gms. | Yes | Small clot and numerous subependymal haemorrhages in right lateral ventricle. | Labour induced on account of albuminuria and high blood pressure. |
| 6 | 50 | 22 | 0 | Frank breech | 7½ mths. | ~~Impacted shoulder~~ Extraction. | F. 1,040 gms. | None | Small clot about size of 1 cm. over lower part of right cerebral hemisphere: no haemorrhage into ventricle. | |

## IV. TENTORIAL TEARS and CEREBRAL HAEMORRHAGE.

These conditions were found in 17 or 24.2% of the cases, and for ease of reference they are tabulated in Table 3. The cases occurring in macerated foetuses have not been considered on account of their greater liability to tears owing to the autolysis in maceration. From examination of table 3 it will be seen that 4 cases of dural tear were associated with cerebral haemorrhage; 4 cases of dural tear were unassociated with cerebral haemorrhage; 3 cases shewed diffuse blood-stained serum over the base of the brain; 5 cases had intra-ventricular haemorrhages, and one case had a subdural haemorrhage over the right cerebral hemisphere. In 5 cases delivery was by the breech ( 2 of these were primarily breech presentations). The remaining 12 cases were vertex presentations - 2 were delivered by forceps, one by Caesarean section, and the others were spontaneous deliveries.

One (21) of the two primary breech presentations had adequate ante-natal supervision: attempts at external version during the last month of pregnancy had been unsuccessful: she was a primigravida aet 18, and she was induced on the expected date of delivery: the foetus weighed 3,720 grammes. The second breech presentation had no ante-natal supervision, and went into labour prematurely /

prematurely at 7½ months. The 3 cases of secondary breech presentation were primarily - one vertex, and two transverse presentations. The vertex was converted into a breech on account of placenta praevia, and is therefore classed as one of the unavoidable causes of death. The other two cases, however, could have been prevented if adequate ante-natal supervision had been sought, when the malpresentation could have been diagnosed and rectified before labour commenced.

The vertex case which was delivered by Caesarean section, is hardly correctly classified under cerebral injuries. The injury was in a vital part of the spinal cord. One case of "broken neck" is recorded by Eardley Holland in his series of 300 cases.[16] The nature of delivery in this case was difficult forceps. The history, shortly is - patient was admitted in labour, L. O. A. position; membranes ruptured; os 5 fingers dilated; head overlapping very slightly. She was put on twilight sleep, as it was thought that the head would mould through. 24 hours after admission classical Caesarean section was performed for maternal and foetal distress. There was difficulty in delivering the head, as Bandl's ring was contracting down round the neck of the foetus. At the post-mortem examination an incomplete tear of the cruciate ligament and haemorrhage over the anterior /

anterior surface of the spinal cord from the level of the 2nd - 6th cervical vertebrae was found.

The two forceps cases were easy deliveries: the foetuses were both large - 3,900 grammes, length 56 cms, and 3,740 grammes, length 54 cms. The mothers were both elderly primiparae, aet 25 and 31. Labour was long in both cases.

Of the spontaneous vertex deliveries, two cases, 32 and 58, died from asphyxia primarily; case 23 was born on the district before the arrival of the nurses, and the nature of the delivery was not ascertained. The remaining 7 cases were all premature foetuses - 2 of the cases were at 7 months, 3 at $7\frac{1}{2}$ months, and 2 at $7\frac{1}{2}$ - 8 months. F. J. Browne[17] has shewn that the predisposition to cerebral haemorrhage is greatest from 7 - $7\frac{1}{2}$ months and at 8 months liability is somewhat less, but still great, while at $8\frac{1}{2}$ months and over the liability is no greater than at term. The preventability of cerebral haemorrhage is therefore largely dependent on the prevention of the occurrence of premature labour. In cases 5 and 50 the cause of premature labour was not found. Case 10 was syphilitic, and it is recognised that this is one of the frequent causes of premature labour and is also one of the preventible causes. Case 14 was a twin pregnancy. This patient had a history /

history of two previous premature labours - the 1st pregnancy had terminated at the 7th month, and the 2nd pregnancy at the 8th month. The present (3rd) pregnancy terminated at the 7th month. The history was that the pregnancy was normal until 10 days before delivery, when there was a slight vaginal haemorrhage, and a catheter specimen taken at this time shewed a trace of albumen. On examination the day after delivery the blood pressure was $\frac{132}{84}$ mm.Hg., trace of albumen in catheter specimen of urine, blood urea nitrogen 200 mg.% non-protein nitrogen 250 mg.%, and the Wassermann reaction was negative. No history of nephritis was obtained, and there was no oedema found on examination. The case was a district one, and, unfortunately, the husband did not allow the urea concentration test to be carried out. The blood urea and non-protein nitrogen figures are the highest found. They suggest, along with the trace of albumen in the urine, a tentative diagnosis of chronic nephritis, but the woman would require to be under observation for a longer period before one could come to a definite conclusion. Case 25 went into labour prematurely associated with the occurrence of high temperature and influenzal cold. Case 27 was an albuminuria of pregnancy, who was induced on account of the non-improvement of the condition under treatment. Case

47 was a twin pregnancy with hydramnios. The Wassermann re-action was negative. To sum up, only two (10 and 27) and probably a third case (14) might have been prevented by treatment and ante-natal care.

Another point, which has been noted by still-birth workers, comes out in this series, namely, the greater liability to tentorial tears and cerebral haemorrhage among primigravidae. 12 out of the 18 cases were primigravidae; 3 were 2-para; 1 a 5-para, and in the remaining case the parity was not ascertained.

The tendency to intraventricular haemorrhage in place of tearing of the tentorium in premature foetuses is shewn in this series. 5 out of the 7 premature foetuses had intraventricular haemorrhages only, while it did not occur in any of the mature foetuses. The greater friability of the vessels in the subependyma and in the choroid plexus and their consequent inability to stand the strain of congestion is generally given as the reason for this tendency to intraventricular haemorrhage in premature foetuses. Further, the coagulation time of the blood is said to be longer in premature than in mature foetuses.

Two of the cases (5 and 14) shewed numerous haemorrhages in other parts of the body as well as the brain. It has been maintained by some observers that haemorrhagic disease /

disease of the newly born is a factor in the production of intraventricular haemorrhages. Rodda[18] in 1920 described the physiological cycle through which the blood of the newly born passes with regard to its bleeding and clotting times. He shewed that at birth it is normal, then it becomes longer, reaching its peak on the 4th or 5th day, and returning again to normal on the 10th day. Rodda[19] states that 25% of all new born babies with cerebral haemorrhage shew evidence of haemorrhage in other organs of the body also. Warwick[20] examined 200 cases, and in 40% found evidence of intracranial haemorrhages, two-thirds of which were due to haemorrhagic diathesis.

TABLE IV.

Pneumonia Neonatorum.

| No. | Record No. | Age. | Parity. | Duration of Pregnancy. | Nature of Delivery. | State of Lungs. | Symptoms. | Age of child. | Size Weightin grms. Sex. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 8 | 30 | 2 | 9 mths. | Spontaneous. | Copious amount of liquor amnii etc. in alveoli and patchy pneumonia. | Child blue at birth. Nothing further observved. | 12 hrs. | 55 cm. 3,535 M. | District case. |
| 2 | 9 | 21 | 1 | 9 mths. | Craniotomy 2 attempts at forceps delivery at home. On admission to R.M.H. liquor amnii foul smelling. D'C"4"- membranes ruptured 20 hrs. before delivery. | Massive catarrhal pneumonia. Considerable amt. of liquor amnii in alveoli. | Nil. | S.B. | Approx. 53 cm. 3,120 without brain | Previous pregnancy had been induced at 8 mths. |
| 3 | 15 | 30 | 2 | 8 mths. | Spontaneous vertex. Membranes ruptured ? one week before delivery | Alveoli & bronchi filled with liquor amnii epithelial cells and lanugo hair, marked polymorphonuclear leucocytic infiltration & patchy pneumonia. | Blue and feeble from birth. | 4 hrs. | 40 cm. 1,615 F. | District case. Twin pregnancy. |
| 4 | 16 | 28 | 0 | 9 mths. | Labour induced. Bougies. Mid forceps delivery. Difficult case. | Massive haemorrhage polymorphonuclear and mononuclear leucocytic infiltration. | Feeble from birth / gradually became more so: was never cyanosed: no difficulty with respiration noted. | 10 dys. | 51½cm. 2,745 M. | Subdural clot under temporosphenoidal lobe. |

| No. | Record No. | Age. | Parity. | Duration of Pregnancy. | Nature of Delivery. | State of Lungs. | Symptoms. | Age of Child. | Size Weight Sex. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 5 | 26 | 25 | 0 | 9 mths. | Vertex spontaneous. Prolonged labour. Membranes ruptured 20 hrs. before delivery. | Lungs unexpanded: massive pneumonia: considerable amt. of liquor amnii in alveoli & bronchi. | | S.B. | 53 cm. 2,870 M. | |
| 6 | 28 | | - | 7 mths. | Spontaneous. | Catarrhal pneumonia of both lungs. consolidation seen on post-mortem examination. | No history obtained. | 3 dys. | 38 cm. 1,020 M. | District case. |
| 7 | 35 | 30 | 2 | 8 mths. | Face, spontaneous delivery. Membranes ruptured 10 hours before delivery. | Little liquoramnii in alveoli but generalised pneumonia. | | S.B. | 45 cm. 2,150 F. | Anencephalitic foetus. District case. |
| 8 | 37 | 37 | 2 | 7 mths. | Vertex Spontaneous. | Catarrhal pneumonia of both lungs - consolidation seen on post-mortem examination. | Child failed from birth. | 7 dys. | 44 cm. 1,460 F. | Albuminuria cause of prematurity |
| 9 | 40 | 27 | 1 | 7 mths. | Spontaneous Breech (L.S.A.) | Massive haemorrhages and pneumonia. | Child feeble from birth, always pale and respirations feeble. | 7 dys. | 37 cm. 900 F. | Twin pregnancy. |
| 10 | 54 | | | 8 mths. | Spontaneous. Vertex. | Very small amount of liquor amnii in alveoli. Massive pneumonia. | | 36 hrs. | 46 cm. 2,140 F. | District case. There is a generalised enlargement of lymphoid tissue - except thymus. |

| Record No. | Age. | Parity. | Duration of Pregnancy. | Nature of Delivery. | State of Lungs. | Symptoms. | Age of child. | Size Weight Sex. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|
| 63 | 40 | 6 | 9 mths. | Vertex Spontaneous. | Patchy pneumonia. | Marked cyanosis from birth. | 18 hrs. | 51 cm. 2,650 M. | Twin pregnancy. |

"it would be necessary to be certain that the child had "not been subjected to any condition which might have "caused pneumonia, i.e., infection of infected secretion "during labour, insufficient covering after birth, etc. "It is well known that pneumonia in young children may "supervene with little or no warning, and may pass to a "fatal termination with great rapidity".

The various modes of infection leading to pneumonia are as follows :

1. Infection from the mother through the placenta - "diaplacental" infection.
2. Aspiration pneumonia, including aspiration of (a) contents of a pathologically infected uterus and vagina; (b) secretion of a normal birth canal; and (c) food or secretion in the nose or mouth of the baby, aspirated after birth.
3. Aerogenous infection, after birth.
4. Metastatic pneumonia, as from a primary infection of the navel or intestine.

Hess Thaysen[24] attributed most of the infections in his series to aspiration of virulent organisms from the birth canals of apparently normal mothers. He did not believe that the pneumonia began in utero, from aspiration of infected amniotic fluid; but it is to be noted that he did not examine the placentae and membranes in these cases, and also that his autopsies did not include microscopic examination of still-births.

F. J. Browne[25] found pneumonia in four still-born foetuses /

foetuses and 24 neo-natal deaths in his second series of 200 cases; he noted the association of prolonged rupture of the membranes and pneumonia, and concluded that the infection occurred by infection of the amniotic cavity by organisms in the vagina or cervix or introduced from without on fingers or instruments, the organisms being inhaled by the foetus during premature inspiratory efforts. Browne also noted the large proportion occurring in prematures, and concluded that the premature infant is about 14 times as liable to die from pneumonia as the infant born at full term.

Johnson and Meyer[26] investigated the cases of still-births and neo-natal deaths shewing evidences of pneumonia at the Sloane Hospital, New York. In the series of 500 cases they found evidence of pneumonia in 97 or 19.4%: 30 of these occurred in still-born foetuses. They concluded that the great majority of cases of congenital pneumonia are apparently due to aspiration of infected amniotic fluid following infection of the amniotic sac after premature rupture of the membranes.

The three cases of pneumonia in still-born foetuses in this series were all associated with premature rupture of the membranes. In two of the cases the presentation was vertex, and membranes were ruptured in each case 20 hours before delivery. One of the cases (9) had labour terminated /

terminated by craniotomy. The third case was a face presentation, in an anencephalic foetus where the membranes were ruptured 10 hours before delivery. The delivery was spontaneous. They were all mature foetuses.

In 3 of the 5 neo-natal deaths, where the infection was probably ante-natal, a note was made at birth that the infant was markedly cyanosed: these three cases all died within 18 hours of birth - 1 aet 4 hours, 1 aet 12 hours and one aet 18 hours. The 2 cases where no note of anything unusual in the appearance of the infant was made were born on the district, and they were both premature infants, one at the 7th and the other at the 8th month, and died aet 36 hours and 3 days. The findings in these cases of still-births, as well as the cases of Johnson and Meyer and F. J. Browne, disproves Hess Thaysen's statement that infection does not occur in utero, while the three cases with marked cyanoses at birth, and remaining so until death, is also suggestive that ante-natal infection may be the cause of neo-natal death. The remaining neo-natal case lived for 7 days, The infection was probably neo-natal: The child had been noted to fail from birth, but there was no history of cyanosis.

Pathology :

Naked eye examination of the lungs was not of much value /

value, evidence being found in only 3 cases. The disease may originate in the terminal bronchioles or bronchi or may arise through the distribution of bacteria into the lung by way of the blood stream. It seems worth while to give here the distinguishing features between lobular and lobar pneumonia, as described by noted authorities. Green[27] says "lobular may be distinguish-"ed from lobar by (1) the fibrin network and exudation "of red blood corpuscles is not nearly so pronounced; "and (2) there are present in the alveoli and bronchioles "considerable numbers of large, clear, round swollen "cells with a round or oval nucleus, probably represent-"ing the desquamated epithelial cells of the bronchioles. "The exudation in broncho-pneumonia is of a serous nat-"ure, contains a certain amount of mucus, and a large "number of polymorphs, which become more abundant as the "process proceeds". In his description of lobar pneumonia he says : "in some cases, the disease may affect "scattered areas in both lungs, being lobular instead of "lobar in distribution. This form is frequently seen "in children x x x The inflammation of the lung "is always accompanied by an inflammation of the pleura "and the bronchi are always inflammed". Muir[28] in his description of broncho-pneumonia says "fibrinous plugs "are not infrequently found in the air vesicles around "an /

"an inflammed bronchus but apart from this situation "fibrin formation is uncommon" and lays great stress on the "presence of a fibrinous reticulum filling the "alveoli" in his description of lobar pneumonia. Beattie and Dickson give very similar descriptions to those of Muir in their textbook on Special Pathology. McCallum[29] gives the various stages in the development of broncho-pneumonia thus : "intense inflammation of the larynx, "trachea and larger bronchi extends into the terminal "bronchioles, where in the same way an outpouring of an "exudate rich in leucocytes and red blood corpuscles "occurs This fills the terminal and neighbouring alveoli. "Desquamated epithelial cells and a network of fibrin "are added, and the alveoli thus contain plugs of exudate "more or less consolidated by the filaments of fibrin. "The old distinction between broncho-pneumonia and lobar "pneumonia does not seem to me accurate, since there is "commonly a great deal of fibrin in the exudate in the "broncho-pneumonic patches, particularly in their more "distal portions".

The features described by Johnson and Meyer in their study of pneumonia in the still-born and the new-born were "the diffuseness or wide extent of the inflammation, "which may involve fairly uniformly practically all of "the alveoli throughout the whole lung. The exudate "within /

"within the alveoli consists of a small or moderate "number of polymorpho-nuclear leucocytes with a few "mononuclears, and a variable number of red cells. "Fibrin is usually absent, or present in minimal amounts". F. J. Browne says "granular debris was the main content "of the alveoli, but some endothelial cells (containing "pigment) a few polymorphs, lymphocytes and an occasional "disintegrating red blood cell could still be made out. "At other parts no cells could be made out - the whole "was a disintegrating mass".

Three of the eight cases in this series had a patchy pneumonia, and the remaining five cases a massive pneumonia. In the three cases of patchy pneumonia the disease was lobular in distribution. The bronchioles affected were inflamed, the walls were hyperaemic and infiltrated with leucocytes, while the epithelial cells were swollen and oedematous, and the lumen contained desquamated cells and exudate; The surrounding alveoli contained catarrhal cells, a few red blood cells and mono-nuclear and polymorpho-nuclear leucocytes. Fibrin was not present in the majority of the cases, but in one case it was present in considerable amount. The five cases of massive pneumonia shewed the same features but generalized throughout the whole lung. One feature which was found in the three still-born foetuses and in the

case /

case who died 4 hours after birth was a considerable amount of liquor amnii and squamous epithelial cells. This association has also been noted by Johnson and Meyer and by Browne. The presence of liquor amnii in the lungs is an indication of foetal asphyxia. It was found in several of the cases where death was due to asphyxia in this series, and further in two of these asphyxia cases I found in the sections a leucocytic infiltration in the neighbourhood of one or two of the bronchioles. A leucocytic infiltration is a response to an "irritation". The first stage in the development of broncho-pneumonia according to McCallum is an outpouring of an exudate rich in leucocytes and red blood corpuscles. Further the association of prolonged rupture of the membranes in 5 of these cases makes the probability of infection of the liquor amnii more possible. It seems reasonable, therefore, to conclude that the pneumonia is caused by the aspiration of liquor amnii into the bronchi, and that the type of pneumonia produced is broncho-pneumonia.

Johnson and Meyer examined the placenta and membranes in 13 out of 30 cases of pneumonia in still-born foetuses and found 11 acutely inflamed. They examined the placenta, membranes and cord of 38 cases of pneumonia where death occurred within 3 days of birth and found acute inflammation in 6.

I /

I examined the placentae and cords of several of these cases, but was unable to confirm these findings. The cord findings may have been negative because the section was taken from close to the foetal end. Leucocytic infiltration of the chorion was found in a number of the cases, but not specially in cases associated with pneumonia.

Acute haemorrhagic pneumonia :

The two cases in this series correspond exactly with the clinical and pathological findings usually described. The one case was in a full-time infant, and the other was a premature (7 months) infant. Both these infants were stated to have been feeble from birth, but neither were cyanosed. Death in each case was associated with marked pallor of the mucous membrane. On naked eye examination the lungs were solid and congested. Microscopically the alveoli were either ruptured by or filled with red blood corpuscles, and the blood vessels were intensely congested. There were catarrhal cells in the alveoli and a few polymorphs. The blood appeared to be fresh and the appearance of the section suggested a sudden, intense infection and congestion.

Extravasation of blood into the lungs, not associated with pneumonia, was found in three other cases in this /

this series. two of these being syphilitic premature infants.

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TABLE V.

| No. | Record no. | Age. | Parity | Duration of pregnancy. | Signs of syphilis in mother. | Antenatal care. | Signs of syphilis in the child. | Age of child. | Sex and weight. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 10 | 24 | 0 | 7½ to 8 months | Hard sore Wassermann +++ | N.A.B. | Chondroepiphysitis Periportal cirrhosis. Fibrosis of pancreas. Alveolar thickening of lung. | 36 hrs. | F. 1,440 | Wassermann reaction strongly positive. Intraventricular haemorrhage primary cause of death. |
| 2 | 12 | 27 | 1 | 9 months | None | None | Spirochaetes +++ in foetal organs. | macerated. | F. 3,000 | Wassermann reaction strongly positive. |
| 3 | 24 | 29 | 1 | 9 months | None | None | Periportal cirrhosis. Fibrosis of pancreas. Thickening of lung. etc. | S.B. | F. 3,500 without brains | Hydrocephalic foetus. |
| 4 | 36 | 23 | 1 | 7 months | None | None | Spirochaetes +++ in foetal organs. | 36 hrs. | F. 2,030 | Maternal Wassermann reaction +++ |
| 5 | 39 | 37 | 0 | 9 months | None | None | Typical syphilitic changes in placenta and cord. | S.B. | M. 4,050 | Cause of death - tear of cruciate ligament. |
| 6 | 51 | 28 | 0 | 7½ mths. | Deafness Mental deficiency | Inadequate | Placenta, cord and epiphysis shewed typical changes | macerated | M. 1,100 | Twin of No. 52 |
| 7 | 52 | 28 | 0 | 7½ mths. | Deafness Mental deficiency | Inadequate. | Placenta, cord and epiphysis. | macerated | M. 1,200 | Twin of No. 51 |
| 8 | ~~61~~ 57 | 36 35 | 6 | 7 mths. | Gumma of thyroid gland. | N.A.B. | Spirochaete + | macerated. | M. 810 | Mother died of acute yellow atrophy. |

| No. | Record No. | Age. | Parity. | Duration of pregnancy. | Signs of Syphilis in mother. | Antenatal care. | Signs of syphilis in the child. | Age of child. | Sex & weight. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 9 | 61 | 36 | 7 | 7½ mths. | Wassermann reaction +++ | None | Spirochaete + | macer-ated. | M. 1,700 | |
| 10 | 64 | 27 | 1 | 7½ mths. | None | None | Marked secondary evidences. | 2 days. | M. 740 | |
| 11 | 67 | 38 | 12 | 9 months | None | None | Spirochaete + | S.B. | M. 2,400 | Hydrocephalous and spina bifida. |
| 12 | 70 | 35 | 3 | 7 months | Wassermann reaction +++ | N.A.B. | Epiphysitis Placental changes. | macer-ated. | M. 570 | |

## VI. S Y P H I L I S.

Syphilis was a factor in the cause of death in 12 or 16% of the cases. 6 of these have been fully described under maceration. The remaining 6 cases were fresh foetuses, and in 4 of these (24, 36, 64 and 67) the primary cause of death was syphilis, while in cases 10 and 39 the primary cause of death was intraventricular haemorrhage, and tear of the cervical cruciate ligament, respectively. 3 of the foetuses were premature, (10, 36 and 64); two had hydrocephalous, and one was a full-time foetus. One foetus (36) was born with a well developed syphilitic pemphigus.

It is useful to compare those findings with those of other observers. Eardley Holland[30] found syphilis (certain, probable and possible) the cause of death in 15.3% of cases in 300 investigated, and Whitridge Williams[31] 12.8% in 273 cases. Eardley Holland and Janet Lane-Claypton[32] in a survey of 114 cases of syphilis occurring in 1,673 cases of dead births and neo-natal deaths found that 78 were macerated and 36 fresh foetuses. They classified the past obstetric histories of the multiparae and found out of 170 pregnancies in cases of certain syphilis 49, or 28.7% terminated in dead births or abortions, and in 22 pregnancies in cases of probable syphilis /

syphilis, 13 or 59.1% terminated in dead births or abortions. A further point noted in this report was that the "number of small families is considerably greater in "the cases of probable syphilis than it is among the cases of cer- "tain syphilis. Hence, the possibility that the less "well marked signs in cases of probable syphilis were "due to a gradual weakening of the virulence of the in- "fection is not supported, but rather the contrary". In the present series there were 5 cases of certain syphilis (12, 36, 57, 61 and 67) Cases 12 and 36 were 1-para but both were comparatively young. Case 57 was a 6-para; case 61 a 7-para, and case 67 a 12-para, and their ages were between 33 and 38 years. The cases of probable syphilis were, with 2 exceptions, in patients under 30 years of age, therefore the application of the above data cannot justifiably be applied as the obstetric histories of many of the women in the probable syphilitic group are presumably incomplete.

C. J. Thomson[33] noted in her series of 100 cases that 15 out of the 17 syphilitic foetuses were prèmature. In this series 9 out of the 12 cases were premature, taking 48 cms and under in length as indicating a premature foetus. These figures, appear to one only to be of value if one considers the amount of anti-syphilitic treatment given before and/or during the pregnancy. No note /

note of this is made in Thomson's series. In the present series the only 2 cases which had anti-syphilitic treatment had premature foetuses. Case 10 had a history of syphilitic infection ( primary sore etc.) in the early part of the present pregnancy; and the onset of premature labour was associated with an attack of influenzal bronchitis. Case 57 developed acute yellow atrophy of the liver and the toxins of this condition were most probably the cause of the premature termination of the pregnancyIn these cases it might, with justification be presumed that the pregnancy terminated prematurely owing to complications other than syphilis. An investigation of the case histories of the 7 untreated cases shews that no other cause than syphilis was found for the early ending of the pregnancy, and therefore 58% of the syphilitic pregnancies ended in premature labours. The investigation of the Wassermann re-action on 2,000 out-patients shewed that 4.1% had strong positive re-actions (3.8% strongly positive and 0.3% moderately strong positive), and 0.7% had signs of syphilis although the Wassermann reaction was negative. Therefore taking 4.8 as the percentage of syphilitic pregnant women in Edinburgh, then roughly 2.5% of the cases of premature termination of pregnancy are due to syphilis. F.J.Browne investigated the statistics over a period of two years

at /

at the Royal Maternity Hospital, Edinburgh, and found that 1 out of every 13 pregnancies ended prematurely, i.e., 7.6%. This investigation shews that syphilis has not the important place it was once considered to have in the causation of prematurity.

J. M. Cruickshank[34] found that 32.54% of the viable foetuses of syphilitic mothers were premature, and of these, no less than 68.75% were still-born. He gives the high figure of 10 as the percentage of syphilitic pregnant women. These figures are based on materials obtained from, and clinical records of, 1,000 cases.

3 out of the 7 untreated cases in this series were macerated and deducting these the percentage of viable foetuses of syphilitic mothers is 33.3, which corresponds very closely with Cruickshank's figures.

The classical tests used in the diagnosis of syphilis have been given under the section on maceration. There it was pointed out that in fresh foetuses we have the valuable additional aid of the histological examination of the foetal organs, with the exception of this test the other tests will only be referred to briefly.

Spirochaetes :

Spirochaetes were found in the liver, lungs and spleen in 2 cases (36 and 67). In both these cases the maternal Wassermann reaction was strongly positive, and

no /

no anti-syphilitic treatment had been received.

Maternal Wassermann Reaction :

The reaction was carried out in all except one case. In 4 out of the 5 cases it was strongly positive, and in one case (24) it was negative. The Wassermann reaction was not carried out in case 39 as the mother died the day after delivery.

History or signs of syphilitic infection :

One case (10) was admitted to the hospital for treatment on account of primary sore, inguinal adenitis and generalized rash. This was the only case in which a history of signs of infection were obtained; the others either had not observed the signs, or deliberately concealed their knowledge. Case 36 is a particularly striking example of this fact. One would have thought that surely in this case a history would be obtained, but its absence is in agreement with one's usual findings. Nearly 100 years ago Abraham Colles[35] wrote on this subject "she had never complained of any "symptoms which might lead even to a suspicion of her "having had primary symptoms; nor has any appearance "taken place in her which can even bear a resemblance "to secondary symptoms". An investigation of the findings of numerous observers shews that the majority believe that the symptoms and signs are frequently so slight /

slight as either not to be observed by the patient, or to be observed and soon forgotten. F. J. Browne's [36] conclusions from the examination of 100 cases were : "In cases of old-standing syphilis in multiparae a his- "tory of infection or of primary and secondary manifesta- "tions is rarely given, while in primigravidae signs of "syplilis are usually present at the time of examination "(70%). When such evidences are not present in a primi- "gravida, she is usually elderly with old-standing in- "fection. As every multipara has been a primigravida "at some period, this seems to prove that the reason why "a history is not found in multiparae is not that it has "never been present, but that it has been forgotten or "is knowingly denied". A history or signs of infection were only present in 64 or 53% of the 120 cases which I investigated, when considering the value of the Wasser- mann re-action.

Obstetric history of the mother :

Cases 10 and 39 were primigravidae, and therefore do not come into this section. Cases 24, 36 and 64 were 1-para. Case 24 was aet 29. There was an inter- val of 3½ years between the present and the last preg- nancy. The child of the previous pregnancy is now alive and well. The present pregnancy went to full term and a hydrocephalic female foetus was delivered after craniotomy /

craniotomy. There was no spina bifida, and the destruction of the brain tissue made it impossible to tell the nature of the hydrocephalous. J. Fraser[37] has shewn that in a large proportion of cases of hydrocephalous syphilis is the cause, and "occurs most frequently in "the child, who otherwise does not shew the general "stigmata of the disease - in fact it would appear, "that it is in the ill-defined types of the disease that "the meningeal changes are most in evidence". This case is analogous. The weight ratios of the foetal organs were within the normal limits, and the maternal Wassermann re-action was negative. A diagnosis was made on the histological findings in the placenta, umbilical cord, liver, epiphyses, etc. Cases 36 and 64 had each a miscarriage at the 4th month about one year before the present pregnancy. In both these cases the signs of syphilis were well marked in the foetus, and the maternal Wassermann re-action was triple positive.

The remaining case (67) had what is usually given as a typically syphilitic obstetric history. The 1st pregnancy terminated at full-time in a spontaneous delivery, child lived one hour; 2nd pregnancy terminated at the 7th month, child still-born; 3rd pregnancy full-time, child died aet 3 weeks; 4th, 5th and 6th pregnancies terminated at full-time, children alive and well; 7th and /

and 8th pregnancies terminated at full time, children still-born; 9th and 10th pregnancies terminated at the 8th month, children died aet about 1 month; 11th pregnancy terminated at full-time, child alive and well. The present pregnancy terminated at the 8th month with a still-born spirochaete-positive foetus with hydrocephalous and spina bifida. The maternal Wassermann reaction was strongly positive.

The only case in this series with an obstetric history shewed what is regarded as evidence of infection. A survey of the literature shews that the obstetric history is frequently of considerable value in the detection of syphilis. The relationship between the date of infection and the occurrence of miscarriages, premature and mature labours and still-births, was discussed under the section on maceration.

External appearances of the child suggestive of Syphilis :

One case (36) was born with a well-developed rash. The full description is given in the case records. The occurrence of a rash on a syphilitic baby is not common within the first four weeks of life. It is usually seen on the plantar aspects of the feet. I investigated the case histories of 80 syphilitic babies and found a record of syphilitic pemphigus in only 3 cases. These developed /

developed on the 3rd day after birth, and were only slight. J. Thomson [38] says that syphilitic pemphigus generally occurs from the 4th to the 6th week of life, therefore more cases may have occurred in the 80 cases investigated, as they were usually discharged from hospital on the 10th day.

Four of the six fresh foetuses were premature, and two were hydrocephalic. The significance of these two features has already been pointed out. Jaundice was not found in any of the cases.

Liver :

The frequency distribution of the weight ratios of 37 non-syphilitic fresh foetuses is as follows :

| Period of development | 15.1 - 20 | 20.1 - 25 | 25.1 & over |
|---|---|---|---|
| 39 weeks and over | 6 | 9 | 5 |
| 34 - 38 weeks | 4 | 1 | - |
| Below 34 weeks | 6 | 6 | - |

and the frequency distribution of the weight ratios of 6 syphilitic fresh foetuses is as follows :

| Period of development | Below 15 | 15.1 - 20 | 20.1 - 25 | 25.1 etc. |
|---|---|---|---|---|
| 39 weeks and over | - | 1 | 1 | - |
| 34 - 38 weeks | - | 1 | 1 | - |
| Below 34 weeks | 1 | - | - | 1 |

It will be seen that the lowest weight ratios in the non-syphilitic group were between 15.1 and 20, the actual figure being 15.2 which occurred in 2 cases. The highest weight ratio was 13.6. In the syphilitic group it will /

will be seen that there is one weight ratio below 15 and one above 25 in the period of development below 34 weeks. The latter was found in the most extensively diseased case of the series. It may be remembered that there were two weight ratios below 15 in the 6 macerated foetuses (cases 12 and 57) The liver rate ratios in these cases were 14.6 and 11.5. The heaviest weight ratio in a normal foetus found by F. J. Browne[39] was 12.5, while 27 out of 31 cases of certain and possible syphilis in his series gave a weight ratio under 12.5. He therefore concluded that 12.5 could be considered as the lowest normal weight ratio, and that a ratio below this indicated probable syphilis. Eardley Holland[40] found the mean liver weight ratio of 162 fresh foetuses to be 21. The mean liver weight ratio of the 37 cases in this series is 21.5. Eardley Holland considered that any fresh foetus with a ratio of less than 15 has an exceptionally heavy liver. He found 7 such cases in his series and from investigation concluded that there is no co-relation between enlarged liver and other pathological conditions. In favour of this view 9 out of the 12 syphilitic cases had a weight ratio higher than 12.5 and one weight ratio of 25.4 occurred in a spirochaete-positive foetus. The 3 weight ratios under 15 all occurred in syphilitic foetuses. One is therefore inclined to view /

view a weight ratio below 15 with suspicion. The margin of difference between the weight ratio of normal and syphilitic cases is small, and a large percentage of the cases fall into the 15.1 - 25 group.

Naked eye examination of the liver yielded negative results. Only one case (24) was thought to be slightly tough on sectioning. Microscopic examination revealed a periportal cirrhosis in all cases, and in addition a fine cirrhosis in two cases (24 and 64).

Spleen :

The frequency distribution of the weight ratios of spleens of 36 non-syphilitic fresh foetuses is as follows:

| Period of development | 200 to 250 | 251 to 300 | 301 to 350 | 351 to 400 | 401 to 450 | 451 to 500 | 501 and over |
|---|---|---|---|---|---|---|---|
| 39 wks & over | 3 | 6 | 4 | 3 | 1 | 1 | 1 |
| 34 - 38 weeks | 1 | 2 | 1 | 1 | - | - | 2 |
| 34 wks & under | - | 2 | 1 | 2 | 2 | 1 | 2 |

and the frequency distribution of weight ratios of spleens of 6 syphilitic foetuses is as follows :

| Period of development | 200 & under | 201 to 250 | 251 to 300 | 301 to 350 | 351 to 400 | 401 to 450 | 451 to 500 | 501 & over |
|---|---|---|---|---|---|---|---|---|
| 39 wks & over | - | 1 | 1 | - | - | - | - | - |
| 34 - 38 wks | 1 | 1 | - | - | - | - | - | 1 |
| 34 wks & under | 1 | - | - | - | - | - | - | - |

A study of the above reveals that the lowest normal weight ratios in this series were between 200 and 250 and that 50% of the normals were between 250 and 300. The lowest normal ratio was 212 in case 46, and the highest /

highest normal ratio 1,333 in case 35; there is therefore a wide range of normal ratios. Comparing this with the syphilitic ratios two cases are under 200, two cases are in the 200 - 250 group. and 2 cases above 250. The two low ratios occurred in cases 36 and 64 - the ratios being 135 and 98.7 respectively. Case 36 had the highest liver weight ratio of the syphilitic group, and case 64 the lowest. The two cases with spleen weight ratios between 200 and 250 were cases 24 and 67, the ratios being 219 and 218 respectively, and their liver weight ratios were the next lowest to that of case 64. There is, therefore, an association between the ratios of liver and spleen in a majority of these cases.

Taylor and Forrest-Smith[41] concluded that any dead-born foetus with a spleen exceeding 0.45% of the body weight, associated with either a positive Wassermann reaction or a probable osteo-chondritis, should be considered as having died of syphilis.

F. J. Browne[42] concluded from his investigation that it was justifiable to fix a ratio of 150 as the heaviest normal, and found that the spleen of the syphilitic foetus is much more frequently abnormally enlarged than the liver, and that splenic enlargement is an important aid in the diagnosis of syphilis in the absence of the spirochaete.

From /

From this series of cases one is inclined to think that the ratio of 150 is too low, and to agree with Taylor and Forrest-Smith's figure of 0.45% of the body weight, i.e., a ratio of 222. The ratio of 4 out of the 6 cases in this series was below 222, and only 2 of the 6 below 150. Further, the lowest normal in this series was 212 and therefore one considers that a ratio of not less than 200 should be taken as the lowest normal.

The highest spleen weight ratio among the syphilitic foetuses was 570, and it occurred in case 10 where the liver ratio was 24. The mother of this case had a course of N.A.B. treatment (4.5 grammes) during the last 3 months of her pregnancy. This may account for the less well defined signs of syphilis found in this foetus.

Visible enlargement of the spleen was seen in 4 of the 6 cases: the largest spleen was in case 36, and weighed 15 grammes, weight ratio 135. In this case also there was a marked perisplenitis with adhesions to the splenic flexure of the colon. This was the only case of peritonitis in the series. The actual proliferative and degenerative changes were not well marked on microscopic examination. The spleen pulp was hyperaemic in all the cases, but this is found in non-syphilitic cases. The changes in the Malpighian bodies were in many cases indefinite, some were poorly nourished and fibrotic /

fibrotic - e.g., case 36, and to a less extent in case 64. Case 36 had in addition marked proliferation of the capsule, with hyperaemia and lymphocytic infiltration.

Pancreas :

Neither the weight nor the naked eye examination were of any value in the diagnosis of syphilis. The variation in the weight of the pancreas was between 0.75 and 4 grammes. With such a small margin it is hardly to be expected that the weight ratio would be of assistance. On section in four of the cases the pancreas was noted as tough, but this was noted in 2 non-syphilitic cases. Microscopic examination was more helpful. 3 of the cases had a very definite increase in the fibrous tissue between the acini. In none of the cases examined was there any increase in the number of the Islets of Langerhans.

Kidneys and Suprarenals :

The weight ratios of both these organs were found to be of no assistance as they were within the limits found in normal foetuses. Naked eye and microscopic examination were also of no assistance. The points looked for on microscopic examination were haemorrhages, particularly in the medulla, and degenerative changes in the vessel walls, but no evidence of these conditions was seen.

Thymus :

Thymus :

The mean of the weight ratios of thymuses of 39 fresh non-syphilitic foetuses was 413. The mean ratio of thymuses of 6 fresh syphilitic foetuses was 471. This slight increase in the weight ratio of syphilitic foetuses has been noted by Holland and other workers, but it cannot be regarded as of diagnostic value as there is not infrequently a marked deviation from the normal in non-syphilitic foetuses.

No unusual feature was noted on naked eye examination of any of the thymuses. Microscopic examination however, revealed fibrosis in several of the cases. The case with most marked changes was 36. Hassal's corpuscles were more numerous than normal and were scattered throughout the substance of the gland; each corpuscle was larger than normal, and in many instances were replaced by polymorphs. There was also a definite increase in the reticulum cells and diminution in the number of lymphocytes. These features were also shewn, but in a less degree, in cases 10 and 64.

Thyroid : The weight ratios were again of no assistance for a similar reason to that given for the pancreas. Naked eye examination was equally unavailing. On microscopic examination a marked fibrosis

was /

was found in one case (64). The fibrous tissue had caused destruction of the acini, and some of the remaining acini contained little or no colloid. The lumen of a few other acini were filled with cubical epithelial cells. Two cases (10 and 36) shewed destruction and vacuolation of the cells in the acini and little colloid in the lumen. A considerable amount of work has been done on the thyroid gland in the full-time foetus and in the newly born, and I. Murray[43] in a recent article has shewn that vacuolation and disappearance of colloid is due to post-mortem degeneration. The only evidence of syphilis, therefore, is fibrosis.

Lungs :

Only one case (36) shewed evidence of pneumonia alba on naked eye examination. It was present in both lungs, but most marked in the right lung. The lungs were tough on section, and of a yellowish-white patchy colour. Microscopic examination shewed thickening of the pleura, of the alveolar wall with destruction of a large number of alveoli, diminished blood supply and increase in the fibrous tissue around the bronchi and interlobular septa. Cases 10, 64 and 67 shewed these features, but to a less marked extent. In addition cases 10 and 24 shewed extensive interstitial haemorrhages which were the /

the most extensive seen in this series. Extensive interstitial haemorrhages are believed by some writers to be a characteristic feature of syphilis.

Chondro-epiphysitis :

This was visible to the naked eye in one case. 5 cases were examined microscopically and it was found definitely present in 4, and doubtfully in a 5th. Eardley Holland[44] noted osteo-chondritis in 36 out of 37 spirochaete-positive foetuses examined on naked eye examination. F. J. Browne[45] found it in 2 cases in his second series. C. J. Thomson[46] noted it in 5 out of 17 syphilitic cases. Taylor and Forrest-Smith[47] noted it in 18 out of 23 cases. The personal factor as to what counts as irregularity of the epiphyseal line seems to be a factor in the usefulness of this condition as an aid in the diagnosis.

Umbilical Cord :

The umbilical cord was examined in four out of the 6 cases. Case 39 was the most typical section, as there was thickening and marked lymphocytic infiltration of the media and intima of the vein and arteries. Case 24 shewed similar changes, but in a lesser degree, and most marked in the vein. Cases 36 and 67 shewed no deviation from the normal. The section examined was taken from close up to the foetal end of the cord, but in spite of an exhaustive search no spirochaetes were found.

Placenta /

Placenta :

The placenta was obtained in all except one case (10), and this was a neo-natal death. In only one case (64, where it was 3.2) was the weight ratio greater than 4, and in this case the significance is lost as this was a premature foetus. Eardley Holland[48] concluded "heaviness of the placenta is not a sign of "great worth in the diagnosis of foetal syphilis, be- "cause (a) amongst the non-syphilitic placentae, a fair "number are as heavy as, or heavier than, the placentae "of the spirochaete positive foetuses; (b) amongst "the placentae of the spirochaete-positive foetuses, a "fair number are no heavier, or less heavy, than the "placentae of the non-syphilitic foetuses". F. J. Browne[49] concluded "while the placenta may be said to be "practically of no value in the diagnosis of syphilis in "the full-time infant, in the premature infant and in "the macerated foetuses it may afford strong evidence."

Recent work has also shewn that the naked eye appearances of the syphilitic placenta yields generally nothing of note. This statement bears out the findings in this series where no distinctive features were found. Cases 36, 39 and 64 shewed uniform increase in the size of the villi, diminution of the intervillous spaces, diminished blood supply and thickening of the walls of the /

the main bloodvessels on microscopic examination. In case 24 these changes were present to a slight extent. The sections examined of case 67 shewed the appearances of a normal placenta.

Diagnosis of Syphilis :

The detection of the spirochaete pallida is the only certain evidence of syphilis, but the spirochaete is sometimes not found in cases which are definitely syphilitic, and then one is dependent on the so-called secondary signs. These secondary signs are changes found in syphilitic, but not in normal foetuses. The standard adopted by Eardley Holland for the diagnosis of syphilis in the absence of the spirochaete pallida is "the "combination of a positive maternal Wassermann reaction, "with osteo-chondritis or typical placental changes or "still more with both together, may be accepted as sure "evidence of foetal syphilis. Osteo-chondritis by it- "self is generally accepted as sufficient evidence; the "fact that all the examples of osteo-chondritis in this "series occurred in association with other evidence of "syphilis re-affirms the accepted view that it is "pathognomonic".

The naked eye evidence is meagre. 4 out of the 6 cases shewed enlargement of the spleen and one an osteo-chondritis. The microscopic examination, however, is frequently /

frequently of great assistance, e.g., case 39 where the sections of the umbilical cord first led to the suspicion that syphilis was present.

The maternal history, particularly the obstetric history, is frequently of considerable assistance. A history of infection, or signs of infection, are naturally of more value, but they are rarely obtained. The maternal Wassermann re-action ought to be taken in every case; a positive re-action, particularly if obtained on more than one occasion may be taken as diagnostic of syphilis, but a negative re-action does not exclude the diagnosis.

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## VII. P R E M A T U R I T Y.

There are two cases (17 and 29) in this series, where no other cause of death than prematurity was found. The cause of prematurity is related to the cause of premature onset of labour. Our knowledge of the reason for the onset of labour after 40 weeks gestation is still deficient, and although we know that two or three conditions are frequently associated with the onset of premature labour, it is not surprising to find a number of unexplained cases. There are an unusually large percentage of premature foetuses in this series, and it will be of value to consider them all together here, with a view to determining the conditions most frequently associated with the onset of premature labour.

1. Placenta praevia (3 and 7) - - 2 cases
2. General foetal oedema (1 and 62) - 2 "
3. Anencephaly (35) - - - - 1 "
4. Syphilis (10,36,51,52,57,61,64,67 & 70) 9 "
5. Maternal heart disease (43) - - 1 "
6. Twin pregnancy per se (45 and 47) 2 "
   (Twin pregnancy & Albumin-
   ( uria - 14,15,31 & 40 - 4 cases)
   (Twin pregnancy and
   ( Syphilis 51 and 52 - - 2 cases)
7. Albuminuria (6,11,13,14,15,18,25,31,34, 37,38,40,41,43, and 65) 15 "
8. Cause not ascertained (5,17,26,28,29, 30,50,54) - - - 8 "

The large number of cases associated with albuminuria will be noted. This series was in no way selected, but in the first 40 cases there was an unusually

large /

large number of cases of albuminuria. The findings in these cases have been given in full in the case records and in other parts of this Thesis.

Of the 8 cases where the cause of premature labour was not ascertained an exhaustive search by means of a personally taken clinical history, Wassermann re-action, urinary and urea concentration test, were carried out in five of the cases, with negative results. Our knowledge of the cause of premature labour in these cases is as deficient as our knowledge of the cause of the onset of mature labour. A study of the known causes shews that preventive measures could have been applied to 25 out of the 40 cases, namely, those where the cause of death was syphilis, maternal heart disease and albuminuria. Only 8 of the 40 cases had any ante-natal care; 4 of these were syphilitic women who came too late in their pregnancy for treatment to be of any avail; 3 were albuminuric, 2 of whom refused to stay in hospital although advised to do so, and the remaining case who received ante-natal care was delivered of a foetus with general foetal oedema.

These cases impress upon one again the great need there is for training of medical students in the necessity of adequate ante-natal supervision of all pregnancies.

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## VIII. FOETAL STATES.

There are two cases which have not come under any of the previous headings. They were both cases of foetal deformity. Case 48 was an anencephalic and case 62 had general foetal oedema. They are described fully in the case records. There was no associated deformity in either case. The maternal Wassermann re-action was negative, and no history of albuminuria or of previous deformities was obtained. The two cases shewed the typical features of the malformation of which they were examples.

In addition to these cases there were five cases of foetal states where some other cause of death was found. These are :

1. General foetal oedema - Case 1
2. Hydrocephalous Cases 12 and 24
   Hydrocephalous & Spina bifida Case 67
3. Anencephaly - - - - Case 35

Minor deformities found were :

1. Left talipes equino-varus associated with two veins and two arteries in the umbilical cord - Case 26
2. Horse-shoe shaped kidney - Cases 58 & 67 (Case 67 had, in addition, hydrocephalous and Spina bifida).
3. One artery and one vein in the umbilical cord - - - Case 46.

TABLE VI.

| No. of case. | Age of mother. | Parity. | Condition of Foetus. | Presence or absence of oedema | Blood pressure on admission | Albumen on admission | Toxaemia symptoms | Condition of Placenta. | Urea concentration test. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 6 | 37 | 0 | macerated | No oedema | 130/90 | Trace of albumen on one occasion only while in hospital | Antepartum haemorrhage. | Retroplacental haematoma and marked infarction | 11.8.26, i.e. 5 months after delivery. Vol. cc. 120. 60. 70. mg.% urea 2.5 3.15 3.15 | Patient reported at hospital 5 months after delivery - urea concentration test normal, no albuminuria, Blood pressure 126/86 mHg. |
| 11 | 37 | 4 | macerated | No oedema | 122/80 | Trace of albumen for last three months of pregnancy | None. | Avascular and old infarctions | Vol cc. - - - mg.% urea 1.2 1.75 1.5 | 3 previous full time pregnancies, one miscarriage at 3rd month. |
| 13 | 32 | 1 | Fresh still born. | Oedema of ankles for last 3 months of pregnancy | | Albumen ++ on admission | Frontal headaches for 1 wk. previous to admission. Had severe revealed & concealed accidental haemorrhage. | Retroplacental haemorrhages & intraplacental + red infarction | 28.4.26 Vol. cc. 113. 109. 56. mg.% urea 1.65 1.65 2.35 | Had oedema of ankles during last month of last pregnancy - (twin pregnancy) Dr. said due to pressure: no albuminuria: Blood urea N. & N.P.N. 12 & 27 mg.% |

| No. of case. | Age of mother. | Parity | Condition of foetus. | Presence or absence of oedema. | Blood pressure on admission. | Albumen on admission. | Toxaemia symptoms. | Condition of Plancenta. | Urea concentration test. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 14<br>15 | 30 | 2 | 1 S.B.<br>2 neo-natal. | No oedema | 130/80 on day following delivery | Trace of albumen | Slight ante-partum haemorrhage 10 days before delivery | Not obtained. District case. | Not obtained. | 1st pregnancy ended prematurely. 2nd pregnancy normal, full time. Blood urea N. & N.P.N. 200 & 250 mg.% |
| 18 | 27 | 0 | macer-ated. | Oedema of ankles for 8 wks. before admission. | 190/118 | 1.2% | Frontal head-aches for one week before admission. | Numerous small white infarcts. | 27.7.26 i.e. 2 months after delivery Vol. cc. 100 105 63 mg.% urea 2.15 2.55 2.2 | 27.7.26. Faint trace of albumen in catheter specimen. Blood pressure 120/80. Blood urea N. & N.P.N. 11 & 23 mg.% |
| 27 | 39 | 1 | Neo-natal | Oedema of ankles noted 3 weeks before admission | 166/98 | 0.4% | Head-aches and blurring of vision for one day before admission. Labour induced as symptoms did not disappear with treatment | Numerous small infarcts | Vol. cc. 40. 61. 32. mg.% urea 0.95 1.35 1.95 | had albuminuria and high B.P. during last pregnancy for which labour was induced. History of rheumatic fever at age of 21 and of scarlet fever between these 2 pregnancies. Blood urea N. & N.P.N. 27 & 52. mg.% |

| No. of case. | Age of mother. | Parity. | Condition of foetus. | Presence or absence of oedema | Blood pressure on admission. | Albumen on admission. | Toxaemia symptoms. | Condition of Placenta. | Urea concentration test. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 31 & 40 | 27 | 1 | 1 S.B. 2 neonatal. | Oedema for 2 weeks before admission. | 194/100 day following delivery | + + | Frontal headaches for 2 days before delivery. | No obvious infarctions but microscopic examination showed red infarcts. | 25.5.26. Vol. cc. - - - mg.% urea 2.0 2.4 2.5 | Previous pregnancy had ended prematurely, no history of oedema or urinary complication. Blood urea N. & N.P.N. 10 & 24. |
| 39 | 38 | 6 | S.B. | Oedema noted from 3rd month. | 182/110 | + + + | Headaches more severe during last week before admission. | Several haemorrhages into placenta. Numerous red infarcts & 2 old infarcts. | 10th day after delivery. Vol. 66. cc. 95.46.67. mg.% urea 2.5 2.65 2.45 | 1st - 4th pregnancies normal, 5th & 6th pregnancies complicated with albuminuria: ended prematurely. Blood urea N. & N.P.N. 15 & 30 mg.% |
| 37 | 37 | 2 | Neonatal | No oedema | 120/80 | Trace | Slight antepartum haemorrhage. | Retroplacental haematoma | Not obtained | Previous two pregnancies normal. Had amputation of cervix since last pregnancy. Blood urea N. & N.P.N. 10 & 17 mg.% |

| No. of case. | Age of mother. | Parity. | Condition of Foetus. | Presence or absence of oedema. | Blood pressure on admission. | Albumen on admission. | Toxaemia symptoms. | Condition of placenta. | Urea concentration test. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 38 | | 4 | macerated. | Oedema of hands and face 5 days before admission. | Was collapsed on admission to hospital. | + | Antepartum haemorrhage. | Numerous red and white infarcts. | 31.5.26. 10th day after delivery. Vol. cc. 50 110 50 mg.% urea 1.5 1.5 1.8 11.8.26 Vol. cc. 180 85 80 mg.% urea 1.5 1.75 2.15 11.8.26 no albuminuria B.P. 128/90 | 1st & 2nd pregnancies normal, F.T. children alive and well. 3rd pregnancy ended at the birth of a macerated foetus. 4th pregnancy terminated at 4th month. Blood urea N. & N.P.N. 17 & 29 mg.%. |
| 41 | 27 | 4 | macerated | None. | Was collapsed on admission. | + | Antepartum haemorrhage. | Marked infarction and completely separated at time of Caesarean operation. | Patient died in hospital. | 4 previous pregnancies normal F.T., children alive and well. |
| 42 | 38 | 9 | macerated | None | 128/80 | + + | Slight antepartum haemorrhage. | Large retroplacental haematoma: placental surface ploughed up. | Not obtained | 8 previous pregnancies, normal, F.T. |

| No. of case. | Age of mother. | Parity. | Condition of Foetus. | Presence or absence of oedema | Blood pressure on admission. | Albumen on admission | Toxaemia symptoms. | Condition of placenta. | Urea concentration test. | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 45 & 47 | 20 | 0 | 1 macerated 2 born alive. | Oedema of ankles for 1 month before admission. | 128/84 | + | - | Intraplacental haemorrhages and red infarcts. | Not obtained | Blood urea N. & N.P.N. 9 & 19 mg.% |
| 51 & 52 | 28 | 0 | macerated. | None. | 132/82 | None. | None. | No infarcts. No haemorrhages. | 10 days after delivery. Vol. cc. 100 105 105 mg.% urea 1.4 0.95 1.00 | Syphilitic case. Blood urea N. & N.P.N. 12 & 28 mg.% |
| 63 & 66 | 40 | 6 | 1 S.B. 2 born alive. | Oedema since early months of pregnancy. | 146/92 | + + | | Red and white infarcts. | 1 month after delivery Vol. cc. 85 106 66 mg.% urea 1.9 2.45 2.2 | 4th & 6th pregnancies were complicated with albuminuria. Blood urea N. & N.P.N. 16 & 30 mg.% |
| 65 | 28 | 2 | macerated. | no oedema | 198/120 & 186/120 during last month | Trace of albumen during last month of pregnancy. | Frequency of micturition, pain in left side and heartburn. No headaches. | Avascular with red and white infarcts. | Not obtained. | 2 previous pregnancies F.T. $C_1$ a.w. $C_2$ S.B. |

21 foetuses 10 macerated, 5 still-born, 6 born alive.
(5 sets of twins)

16 mothers.

Average age of mothers = 32.2 yrs.

Mean parity = 2.6.

3 cases of recurrent toxaemia, Cases 27, 34 and 63 & 65.

# IX. PROPHYLAXIS.

Before prevention of still-births and neo-natal deaths can be undertaken, the causes must be fully understood. A considerable amount of information can be obtained by investigation of statistics, causes and associated factors of still-births. To determine the cause in each case a clinical and biochemical investigation of the mother, as well as the post-mortem investigation of the foetus, requires to be carried out.

An investigation of statistics shows that the infant mortality rate has fallen 50% in the last 50 years, viz., 149 in 1871 - 1880, and 75 in 1924 (England and Wales), but a closer examination in the period of life which mainly concerns us (the first month of life) shews that there has been no appreciable difference in the infant mortality rate for that period: further, the rate for the first month of life is the same in each quarter of the year, while in older children there is a seasonal difference in mortality. The rate is the same for all classes of the community. Brend[50] quotes the figures of the Medical Officer of Health for Brighton, where the death rate of babies under one week is 20.4 in the poorest classes and 20.5 in the well-to-do classes, and concludes "the great bulk of the deaths are due to "some /

"some obscure internal derangement of normal processes "in the mother or infant, which are either independent "of the external environment or are due to some factor "or factors in the external environment equally common "among all classes and under all circumstances".

The literature on the causation of still-births and neo-natal deaths, is extensive. The more important of the contributions have been recently edited by Eardley Holland and J. Lane-Clayton, and published by the Medical Research Council. The percentage frequency distribution of the causes of still-births given there is :

| | |
|---|---|
| "Pelvic deformity and malformations - | 33.0% |
| "Placenta praevia - - - - | 22.1% |
| "Albuminuria - - - - - | 12.1% |
| "Deformities including hydrocephalous | 11.5% |
| "Syphilis - - - - - - | 9.5% |
| "Premature labour not due to disease of the father and mother- - - | 3.9% |
| "Prolapse or torsion of cord - - | 3.4% |
| "Other general diseases - - - | 2.7% |
| "Other causes - - - - - | 1.1% |
| "Asphyxia at birth - - - - | 0.7% |

A study of this table shews that nearly 50% of these cases might, according to our present knowledge, have been prevented. The occurrence of malpresentation, albuminuria and syphilis might have been prevented by Ante-natal supervision.

1. Ante-natal supervision.

Every woman should be supervised during her pregnancy, as by this means many of the complications of labour /

labour, as well as the toxaemias of pregnancy and subsequent ill-health, can be averted. Ballantyne has shewn that the still-birth rate is reduced by 50% where ante-natal care, and treatment if necessary, is carried out - see table quoted in Introduction, page 2. Supervision should be carried out during the whole of the pregnancy, but there is no need for frequent physical examinations. On the contrary these are rather to be deprecated as they are apt to make the mother nervous and so regard the pregnancy as a pathological instead of a physiological condition. It is now generally agreed that all that is necessary in a normal case can be learned by careful systematic abdominal examination - inspection, palpation, pelvimetry and auscultation, - with one vaginal examination preferably not before the 7th month. The urine should be examined for albumen and sugar once a month in the early months of pregnancy and more frequently in the later months. A routine blood test should be carried out at the first or second visit to the clinic. The patients at the Edinburgh clinic rarely raised any objection to this test, though one realises that it cannot always be carried out in private practice. The value of the routine use of the Wassermann re-action has been shewn elsewhere in this Thesis. /

Thesis. The blood pressure, systolic and diastolic readings should be taken at every visit to the clinic. I investigated the records of 25 cases of albuminuria occurring among the out-patients and found that the first evidence of toxaemia in six cases was raised blood pressure which preceded the appearance of albuminuria by a fortnight in four out of the six cases. In two of the 25 cases, although the blood pressure was $^{180}/_{100}$ mm.Hg. no symptoms were complained of when the patients came to the clinic.

2. Ante-natal Treatment :

General advice regarding diet and hygiene is given to all the patients.

Renal Treatment : The great importance of this subject in relation to still-births is shewn by the large number of cases (see table VI), and also by its place in the list quoted on page opposite 180. The first thing that strikes one when examining the table is the age of the patients; with one exception they are all over 27 years of age, the mean age being 32.2. Further the 3 oldest cases are cases of recurrent toxaemia. It is a significant fact that out of 16 cases there are 3 recurrent toxaemias, and these 3 mothers are the oldest. One is prompted to ask the question "is this the true percentage of recurrent toxaemias in this group ?"

Many /

Many of the younger mothers would, presumably, have later pregnancies. A clinical and biochemical investigation revealed an interesting fact. Case 27, aet 39, gave a history of rheumatic and scarlet fevers, and shewed all the features associated with a chronic nephritis complicating pregnancy: moderately raised blood pressure ($^{166}/_{98}$ mm.Hg.); 0.4% albumen in the urine; gradually progressive oedema for the last two months of pregnancy; non-response to treatment, requiring induction at the 7th month; the blood urea nitrogen and non-protein nitrogen both raised (27 and 52 mg..%) and the urea concentration test below 2. The other two cases shewed all the clinical and biochemical signs of albuminuria of pregnancy. Their ages were 38 and 40 years respectively; both were 6-para; the blood pressure was high $^{182}/_{110}$ in one case and low $^{146}/_{92}$ mm.Hg. in the other case; there was oedema for a considerable time in both cases; both went into premature labour at the 7th month one had toxaemic symptoms (headaches etc.) for the last week of pregnancy; the blood urea nitrogen and non-protein nitrogen were within the normal limits in both cases, 15 & 30 and 16 & 30 mg.% and the urea concentration test was good in both cases. The biochemical findings in these three cases of recurrent toxaemia, throw doubt on the general belief that recurrences are only rarely associated with /

with a true albuminuria of pregnancy.

One clinical sign which the three cases had in common was earlier occurrences of oedema with each successive pregnancy; the differentiation into true albumininria and chronic nephritis was made mainly on the history of scarlet fever and the bio-chemical findings.

The findings in three other mothers are worthy of note. Case 11 and cases 51 & 52 (twins) may be considered together. They both had low blood pressures, no clinical evidence of toxaemia, and low urea concemtration. The foetuses were premature and macerated, and there was no infarction or haemorrhage in the placenta. Case 11 had, in addition, a trace of albumen on several occasions during her pregnancy. Cases 51 & 52 were syphilitic. The low urea concentration test was suggestive of a chronic nephritis, but these patients would require to be under observation for a longer period, and between their pregnancies, before a diagnosis could be made.

The 3rd mother had also a twin pregnancy (cases 14 & 15). Her previous obstetric history was - 1st pregnancy ended prematurely; 2nd pregnancy normal full time, but child is said to have weighed 5 lbs. In the present pregnancy there was no history of toxaemic signs or /

or symptoms except slight vaginal haemorrhage 10 days before going into premature labour at the 7th month. The blood pressure was 130/80 mm.Hg. on the day after delivery; there was a trace of albumen in the urine, and the blood urea nitrogen and non-protein nitrogen were 200 & 250 mg.% This was a district case, and the husband would not allow a urea concentration test to be done. It would have been interesting to have had another blood urea done a few days later to find if the blood urea had fallen. F. J. Browne[51] in his experimental work on rabbits found "the blood urea not infrequently rose to a very "high figure in a short time, and would almost as rapid- "ly drop from this high figure to normal. Even in cases "in which the blood urea was at a high figure and in "which, therefore, there was an acute nephritis present, "albumen might be entirely absent.". In one case of albuminuria at the Royal Maternity Hospital, the blood urea fell from 98 to 12 mg.% in 3 days. Our knowledge of the cause of this rapid alteration in the blood urea is lacking, but these cases shew the necessity of blood urea and urea concentration tests in cases where there is little or no albuminuria. It seems possible from the results found in this investigation that a certain number of cases where the cause of ante-natal death or prematurity has been hitherto classified as "cause unknown" /

unknown" may be due to renal damage, e.g cases 11 and 14 & 15 of this series.

Anti-syphilitic treatment : The syphilitic group, together with the toxaemic group, are the most promising fields for the prevention of still-births. Further, the treatment of congenital syphilis, not only after birth but also indirectly by treatment of the syphilitic pregnant woman, is an important means of combating the spread of this disease. Naturally, the earlier the treatment is commenced in the pregnancy, the more likelihood there is of a healthy, non-syphilitic child being born. Only two of the cases had treatment in this series, neither had adequate treatment, while in one of the cases treatment was not commenced until after intra-uterine death had occurred.

Treatment of other maternal conditions : There is only one case in this series which comes under this heading (case 46). The mother had a mitral lesion, but did not come under observation until the 8th month of pregnancy when she had acute failure of compensation. All cases with heart lesions should be kept under very careful supervision and, if necessary, labour should be induced before term, or Caesarean section performed. The results in this group are frequently unsatisfactory, particularly regarding the child, but there is no doubt that /

that the mother benefits by having ante-natal care and treatment.

Obstetric measures designed to avoid still-birth from difficult labour :

The performance of version in cases of breech presentation, if diagnosed before labour commences, or if seen sufficiently early in labour, since tentorial tears are more common in breech than in vertex presentations.

Application of pads when the presentation is a posterior one, in order to assist anterior rotation. Prolonged labour met with in posterior presentations leads not only to exhaustion of the mother, but also, occasionally to asphyxia of the child.

Induction of premature labour, the need for this having been discovered in the routine ante-natal supervision. This should, for the child's sake preferably be performed not before the 38th week of gestation. If the degree of pelvic contraction is marked, the performance of Caesarean section at term may have to be considered.

Treatment of haemorrhages.

The prevention of accidental haemorrhage in a certain number of cases comes under ante-natal supervision by the early detection of signs of toxaemia, but in a considerable proportion of cases the onset of haemorrhage is sudden and the treatment is then obstetrical. The

percentage of still-births in cases of accidental haemorrhage is high, owing to the frequent separation of the placenta from the placental site when the haemorrhage is severe. The mother's condition is critical, and requires first consideration. In severe cases of accidental haemorrhage Caesarean section, and occasionally hysterectomy is done on account of the mother's condition.

The maternal mortality in cases of placenta praevia is dependent on the effectual compression of the placental site, and this is usually done by (a) packing the vagina and applying a firm abdominal binder; (b)the foetal breech with traction on foetal leg; or (c) traction on the foetal head. The second method is the one most frequently employed, but it has a high foetal mortality. The Edinburgh teaching in cases of central placenta praevia in primigravida is performance of Caesarean section and the foetal mortality is naturally negligible when this is done.

## X. SUMMARY and CONCLUSIONS.

1. The main causes of death in this series were complications of labour, albuminuria, syphilis and pneumonia.

2. Many of these cases might have been prevented if they had received ante natal supervision.

3. Pneumonia and cerebral haemorrhage were the most frequent causes of death in the neo-natal period, particularly in premature foetuses.

4. There were a large number of premature foetuses in this series. It has been shewn that the percentage of syphilitic cases in this group (i.e. premature foetuses) is lower than it is frequently supposed to be.

5. A biochemical investigation of a large number of the cases in this series revealed an otherwise undetected kidney lesion in a few cases where the pregnancy terminated prematurely, and suggested that a similar condition might have been found in some cases in other series where the cause of prematurity has been classed as unknown. One does not mean to imply that all cases of prematurity are due to nephritis, there are two cases in this series where the cause of /

prematurity was not found. A considerable amount of research still requires to be done on the cause of the onset of normal labour before the etiology of premature labour is satisfactorily explained.

6. Biochemical investigation of these cases of ~~recurrent~~ toxaemia showed that two of them were recurrent albuminurias of pregnancy, disproving the generally accepted theory that true albuminuria of pregnancy does not recur in subsequent pregnancies.

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References :

1. Eden & Holland: Manual of Midwifery, 6th edition.

2. Eden, T.W., Jour. Path. & Bact., 1897, iv. part II. p. 265 - 283.

3. Young & Miller. Proceed. Roy. Doc. Med. 1921, XIV. (Sect. of Obst. & Gyn) pp. 247 - 259.

4 & 7 Browne, F. J. Etiology of Accidental Haemorrhage loc. cit.

5. Winter quoted by F. Cook. Observations on the Tox-aemias of pregnancy. Guy's Hosp. Reports 1924, Vol. 74, No. 2.

6. Ley, Gordon. Proceed. Roy. Soc. Med.

8. Holland, Eardley. The Causation of Foetal Death.

9. Muir & Ritchie, Manual of Bacteriology, 2nd edition.

10. Belding, D.L., The Wassermann Test. Am. Jour. of Syph. 1925, Oct. p.764.

11. Browne, F.J. Influence of pregnancy on the Wasser-mann test and the clinical manifestations of syphilis. Jour. Obst. & Gyny. Brit. Emp. 1923, Vol. 30. No. 4 pp. 519 - 540.

12. Boas, Gammeltoft and Sieck. Abst in ? Jour. Amer. Med. Assoc.

13. Quoted in Special Report Series No. 23 p.12.

14. Polak, quoted by J. King, Intrauterine death. Am. Jour. Obst. & Gynec. 1926, XII. No.6.

15. King, J., loc. cit.

16. Holland, Eardley, The Causation of Foetal Death.

17. Browne, F. J., Edin. Med. Journ. 1924, Octr.

18. Rodda quoted by J. W. Bruce. Haemorrhagic disease of the new-born. Therapeutic Gazette 1926, Octr 15th.

19 & 20. Rodda & Warwick, quoted by J. F. Dicks. Intra-cranial haemorrhage of the new-born. Am. Jour. of Obst. & Gynec. 1926 XII. No. 6.

21 /

References (Continued) :

21. Hess Thayser, quoted by Johnson and Meyer (22)

22. Johnson and Meyer. A study of pneumonia in the still-born and new-born. Am. Jour. Obst. & Gynec, 1925. IX. No. 2.

23. Special Report Series No. 109, p.16.

24. Hess Thayser, loc. cit.

25. Browne, F. J., Brit. Med. Journ. 1922, March 25 and Sept 30.

26. Johnson & Meyer, loc cit.

27. Green, Textbook of Pathology (1923)

28. Muir, Textbook of Pathology (1925)

29. McCallum, Textbook of Pathology (1924)

30. Holland, Eardley., The Causation of Foetal death.

31. Whitridge Williams quoted by Eardley Holland.

32. Special Report Series No. 109.

33. Thomson, C. J., The Causation of Foetal Death. Jour. Obst. & Gynec., Brit. Emp. 1926 XXX. 3

34. Cruickshank, J. N., Brit. Med. Jour. 1922, II. 593 - 594.

35. Colles Work edited by McDonnell. New Sydenham Society.

36. Browne, F. J. Jour. Obst. & Gynec., Brit. Emp. XXX. No. 4, p. 539.

37. Fraser, Surgery of Childhood, Vol. 1. p.492.

38. Thomson, Diseases of Childhood.

39. Browne, F. J., Edin Med. Jour. 1924, Oct.

40. Holland, Eardley, Causation of Foetal Death.

41. Taylor & Forrest-Smith, quoted in Special Report Series No. 109, p. 54.

42. Browne, F. J., loc cit.

43. /

References (Continued) :

43. Murray, I. The Thyroid Gland in the Foetus and in the newly born. Brit. Med. Jour. 1927, I. pp. 5 - 8.

44. Holland, Eardley, loc. cit.

45. Browne, F. J. loc. cit.

46. Thomson, C. J. loc. cit.

47. Taylor & Forrest-Smith. Special Report Series No. 109.

48. Holland, Eardley, loc. cit.

49. Browne, F. J., loc. cit.

50. Special Report Series, 1918. No. 10.

51. Browne, F. J., Etiology of Accidental Haemorrhage and Placental Infarction. Brit. Med. Journ. 1926, I. April 17th.

# CASE RECORDS.

CASE 1 :

4 para, aet 36. $C_1$ full time, died age 10/12 years, cause unknown: $C_2$ miscarriage at the 2nd month: $C_3$ and $C_4$ full time, alive and well. Felt quite well for first 6 months of present pregnancy, but during the last 3 months she felt "horrible" - off food, easily tired, heartburn and frequent vomiting. Came to A. N. Clinic on 12/2/26 on account of constipation and pressure symptoms - oedema of ankles, legs and pubes, and pain under left breast. On 1/3/26 albumen was present in urine, and very marked hydramnios along with oedema of legs and abdominal wall was noted. Patient admitted to Ward. Abdominal wall was tense, greatest circumference 42". 10/3/26 went into labour, general anaesthetic given, os found to be fully dilated, membranes ruptured, and large quantity of liquor amnii escaped; vertex engaging. High forceps were applied, and foetus delivered with considerable difficulty.

Wassermann reaction was negative; teeth and fauces were in good condition; mucous membrane appeared healthy; blood count was not done.

Premature macerated oedematous female foetus, weight 2,350 grms, length 45 cms. Oedema generalized, free fluid in peritoneal cavity. Oedema of face was marked and tongue protruded from the mouth.

Microscopically /

Microscopically placenta shewed irregular enlargement of villi, umbilical cord oedematous and vessel walls fibrosed, but no lymphocytic infiltration and no epiphysitis.

Cause of death - general foetal oedema.

CASE 2 :

Primipara aet 34. No antenatal supervision. Felt well. Admitted in labour, breech well engaged, membranes ruptured 36 hours before admission and before onset of true labour pains. No foetal heart heard on admission. Great difficulty in delivering buttocks, breech hook used, both arms extended and brought down with difficulty; head easily delivered. Felt life until 15/3/26. Membranes ruptured and labour commenced at 2.a.m. 16/3/26, delivered at 2 p.m. 17/3/26. Full time fresh male foetus, weight 3,620 grms, length 52 cms. Large haematoma in upper part of left thigh, oblique fracture of left femur at junction of upper and middle third. Marked congestion of all organs but no haemorrhage; lungs were not expanded; small incomplete tear of right tentorium cerebelli. Microscopic examination of lungs - small amount of liquor amnii in alveoli and bronchioles. No pneumonia.

Cause of death - Intrapartum asphyxia from prolonged 2nd stage.

CASE 3 :

3 para aet 30. 1st and 2nd pregnancies terminated at full time, instrumental deliveries, both alive and well. 3rd pregnancy terminated at 4th month. Present pregnancy, no antenatal supervision, normal until 15/3/26 when she had a sudden profuse vaginal haemorrhage; vagina packed by own doctor, packing removed on 17/3/26, after which there was a constant oozing from the vagina, with another severe haemorrhage at 4 p.m. on 18/3/26. Vagina again packed and patient sent into Royal Maternity Hospital. On admission os admitted 2 fingers, and there was a central placenta praevia. External version performed, and leg brought down through hole in placenta. Foetal heart rate was 170 and irregular before manipulations were commenced. Pituitary extract m.iv. given after manipulations, and child delivered spontaneously half an hour later, still-born.

Premature fresh male foetus, weight 1,940 grms, length 42 cms: marked congestion of all organs: several petechial haemorrhages on surface of lungs and at base of heart, and free straw coloured fluid in pleural and pericardial cavities.

Cause of death - Intrapartum asphyxia following version for placenta praevia.

CASE 4. /

CASE 4:

Primipara aet 39. No supervision during pregnancy: felt well up to last 8 weeks, when she noted oedema of legs, but this disappeared each night with rest in bed. On admission urine contained no albumen, and there was no oedema of ankles. Spontaneous delivery, R.O.A. Total duration of labour 17½ hours - 1st stage 15 hours, 2nd stage 1½ hours, 3rd stage ¾ hour.

Full time fresh female foetus, weight 3,680 grms, length 51 cms; marked lividity; no p.m. rigidity. Thorax - Free straw coloured fluid in pericardial and pleural cavities; a few subpleural haemorrhages mainly round roots of lungs; no subepicardial haemorrhage; there was a persistent thyro-glossal duct. Abdomen - marked congestion and subcapsular haemorrhage in right suprarenal. Brain - Marked congestion, but no haemorrhage and no dural tear.

Microscopically - lungs were not expanded; bronchi, bronchioles and a few alveoli were filled with liquor amnii, epidermal cells and lanugo. There was marked congestion of the alveoli, but no pneumonia.

Cause of death - Intrapartum asphyxia.

CASE 5. /

CASE 5 :

Primipara, aet 24. No antenatal supervision; felt quite well during whole of pregnancy; went into labour prematurely on 21/3/26. Cause of premature labour not ascertained. Vertex presentation, spontaneous delivery of a feeble premature child, who lived 4 hours. Child was pale, breathed feebly, and did not cry properly during its life; did not vomit and had no fits.

Premature fresh female foetus, weight 2,070 grms, length 42 cms: marked pallor of lips and finger nails; congestion of internal organs and numerous subpleural and subepicardial petechial haemorrhages. Brain - large blood clot in both ventricles and tearing of ependyma, no dural tear.

Microscopically - numerous haemorrhages in lung and marked congestion of internal organs.

Cause of death - Cerebral haemorrhage. Probably haemorrhagic diathesis is a factor in this case.

CASE 6 :

Primipara aet 37. No antenatal supervision. Pregnancy apparently normal until 18th March, when she had a sudden vaginal bleeding. No oedema of ankles and no toxaemic symptoms. She was transferred to Royal Maternity /

Maternity Hospital same day. On admission Cervix admitted 2 fingers, no placenta felt, but slight oozing of blood. Vagina packed. 19/3/26 Packing removed, no bleeding, quinine and pituitrin induction commenced. 21/3/26 Urine contained a trace of albumen, Blood pressure $130/_{90}$. 22/3/26. No life felt, and no foetal heart heard. 23/3/26. Slight haemorrhage, labour commenced. Vertex presentation, spontaneous delivery of macerated foetus, premature, male, weight 1,770 grms, length 45 cms. Intense congestion of internal organs, numerous petechial haemorrhages in lungs along sulci of heart, and under capsule of liver; free blood stained fluid in pleural and peritoneal cavities; intense congestion of cortical veins over cerebral hemispheres, but no haemorrhage. Both tentorium cerebelli showed complete tears. Placenta - about half of the maternal surface was infarcted. The infarcts were large and small, and mainly old. An area of 8 x 6 cms of the placental surface was ploughed up by old blood clot.

Cause of death - Antepartum asphyxia following placental separation by retro-placental haemorrhage.

Puerperal history - No albumen in urine; no increased blood pressure; vomited urea given for test. Wassermann reaction negative. Reported 11/8/26. Albumen negative, blood pressure $126/_{86}$

| | | Total vol. ccs | mg urea % |
|---|---|---|---|
| Urea concentration test | (1) | 120 | 2.5 |
| | (2) | 60 | 3.15 |
| | (3) | 70 | 3.15 |

CASE 7 :

Primipara, aet 25. Under irregular antenatal supervision for last four months of pregnancy. In Antenatal ward for one week from January 11th to 19th, and belt ordered for visceroptosis. Re-admitted on 17/3/26 on account of cough, pains in chest and abdomen. 22/3/26 felt sick all day, vomited once. 23/3/26, felt better, but complained of dull pain in left side. 25/3/26 Still complaining of vague pain in lower abdomen, cough less troublesome. 10 p.m. Sudden painless haemorrhage, about 4 ozs lost. Placenta felt in lower uterine segment, vagina packed. 28/3/26 Urine trace of albumen, blood-pressure 112/80, no more haemorrhage, slight pains. 29/3/26 Fairly severe vaginal haemorrhage, cervix only admitted 2 fingers, placenta felt covering about half of internal os, no uterine contractions, no foetal heart heard. Vagina packed with kite-tail pack and $\frac{1}{2}$ cc. pituitrin given half hourly for 2 hours, when it was stopped as patient having good pains. Four hours later bleeding through pack was profuse, pack was removed, and as os still only admitted 2 fingers, external version was done and leg pulled through the membranes. Still-born child was delivered spontaneously 5 hours later, Placenta had to be manually removed Wassermann reaction negative.

Full time, slightly macerated male foetus, weight 2,800 /

2,800 grms, length 48 cms. Marked congestion of all internal organs with numerous petechial haemorrhages into lungs, heart, under capsule of thymus and of left suprarenal.

Cause of death - Antepartum asphyxia following partial separation of placenta. Microscopic examination of lungs revealed extensive interstitial haemorrhages in two areas. There was a slight polymorphonuclear leucocytic infiltration, but there was no evidence of pneumonia.

Urea concentration test on 10th day ; (1) 2.25 mg urea %; (2) 3.4 mg urea %; (3) 3.5 mg urea %.

CASE 8 :

2-para, aet 30. District case, history incomplete. No antenatal supervision, pregnancy was said to be normal; labour was not prolonged; no knowledge of when membranes ruptured; vertex presentation, spontaneous delivery. Child was said to be blue birth, nothing was noted about its respirations, no history of fits or convulsions, died when 12 hours old.

Full time fresh male foetus, weight 3,535 grms, length 55 cms. Congestion of all organs and consolidation of lower lobes of lungs. Microscopically - patchy broncho-pneumonia and copious amount of liquor amnii and lanugo hairs in alveoli and bronchi.

Cause of death - broncho-pneumonia.

CASE 9.:

1-para, aet 21. Admitted in labour after two attempts at forceps delivery had failed at home. On admission head above brim, contraction of inlet (D.C.4") Liquor amnii foul smelling, no foetal heart heard, forcep delivery attempted, but unsuccessful - perforation and craniotomy performed. Had attended ante-natal clinic irregularly, last attendance 6 weeks previously. Previous pregnancy had been terminated at the 8th month on account of the contracted pelvis, child alive and well

Full time fresh male foetus, weight 3,120 grms, length 53 cms. Congestion of internal organs well marked and numerous petechial haemorrhages - lungs, heart, posterior wall of thorax and thoracic surface of diaphragm. Microscopically - lungs not aerated, but shewed definite massive catarrhal pneumonia, and a considerable amount of liquor amnii in alveoli.

Cause of death - Broncho-pneumonia.

CASE 10 :

Primipara, aet 24. Admitted 3/2/26 for treatment for labial sore, from Ward 20 R.I.E. History of primary sore on labia in November 1925. On admission Wassermann reaction +++ strongly positive. Discharged from /

from ward on 16/3/26, sores having cleared up with arsenical injections. Re-admitted on 2/4/26 with influenzal bronchitis, T.102, P.124,R.38. Labour pains started in the evening, and delivery was spontaneous without much distress. Child was premature, and very feeble, vomited even when pipette fed, no convulsions, lived 36 hours. Mother had 4.5 gms N.A.B. between 5/2/26 and 26/3/26.

Premature fresh female foetus, weight 1,440 grms, length 41 cms., marked lividity and p.m. rigidity; marked congestion of internal organs, but no haemorrhage except one into cardiac muscle. Brain - Excess of cerebro-spinal fluid, veins on upper surface of cerebrum not congested, haemorrhage into right olfactory bulb, into optic chiasma, on inferior surface of right temporal lobe, and along sylvian fissure; lateral ventricles filled with blood clot (in right ventricle clot measures $2\frac{1}{2}$ x $\frac{3}{4}$ cms) marked ependymal haemorrhage; no haemorrhage into 3rd and 4th ventricles, but these appear dilated.

Microscopic examination gives definite evidence of syphilis, periportal cirrhosis of liver, fibrosis of pancreas, almost total destruction of thyroid, alveolar thickening in lung, chondro-epiphysitis of lower end of femur, enlargement of villi in placenta and diminution of intervillous spaces. Levaditi examination negative.

Cause of death - Cerebral haemorrhage (syphilis).

CASE 11 :

4-para, aet 37. 1st and 3rd pregnancies full-time spontaneous deliveries, children alive and well: 2nd pregnancy terminated at 2nd month after a fall: 4th pregnancy full-time child died of skin disease at age of 8 weeks. Present pregnancy - came to A. N. complaining of discharge on 22/12/25, trace of albumen found in catheter specimen of urine, blood pressure $122/_{80}$. Treated in A.N. Ward from 18/1/26 to 25/1/26 for vulvitis, cystitis and discharging Bartholinian abscess; trace of albumen in catheter specimen of urine on 11/1/26, 18/1/26 and 4/2/26; blood pressure varied between 122/80 and 140/100 mm.Hg. no oedema of hands and feet; no toxaemic symptoms; did not feel life during last 3 weeks of pregnancy; felt a slow movement in the lower abdomen at night when she lay down; she noticed that her clothes were looser, but her general health improved during these three weeks. Previous illnesses - had chorea between ages of 10 and 14 years. No history of scarlet fever, diphtheria or nephritis. 7/4/26. Delivered spontaneously of a premature, much macerated foetus, weight 1,320 grms, length 40 cms. Congestion of all organs, blood stained fluid in pleural, pericardial and peritoneal cavities. The right tentorium cerebelli had an incomplete tear. Placenta - one half of the placental area shewed large infarcts /

infarcts and fibrosis, the other half showed a surface fibrosis only. The cotyledons were not well marked and the depth of the placenta was not more than 1 cm. in parts. Microscopically - placenta shewed a considerable amount of old and recent infarction and well developed endarteritis obliterans in the blood vessels of the terminal villi; no chondro-epiphysitis; no lymphocytic infiltration in umbilical cord.

Cause of death - ante-partum asphyxia from placental fibrosis.

Puerperal history - No albumen in urine during stay in hospital. Blood pressure did not vary much, the highest during puerperium was 136/84. 17/4/26. Urea concentration test :

| | Total vol.ccs | Mg.urea % |
|---|---|---|
| (1) | 90 | 0.75 |
| (2) | 75 | 1.2 |
| (3) | 110 | 1.0 |

CASE 12 :

1-para, aet 27. Admitted to Royal Maternity Hospital on 10/4/26 with head born; patient anaethetized shoulders extracted with difficulty and rest of body with strong supra-pubic pressure. Foetus macerated and abdomen ascitic.

Last pregnancy terminated 3 weeks before term, child born in Royal Maternity Hospital, weight at birth 5 lbs /

5 lbs, now alive and well. Present pregnancy no Antenatal supervision; more than usual amount of vomiting in first three months and in last month. During second last week of pregnancy mother noticed that foetal movements were not so active; during last week of pregnancy no "life" was felt. No history of primary sore, rash or syphilitic manifestation. Wassermann reaction on 18/4/26 strongly positive.

Full time female foetus with well developed maceration (skin peeled off body); weight 3,000 grms, length 50 cms. Head practically severed from body at level of 3rd cervical vertebra; oblique fracture of right humerus; abdomen distended; abdominal cavity filled with blood-stained serum; liver and spleen enlarged; pancreas enlarged and firm; free blood stained fluid in pleural and pericardial cavities; numerous large subpleural haemorrhages - possibly pneumonia alba in left lung. Placenta not enlarged; not pale and greasy; cotyledons not well-marked; placental surface thickened from maternal to foetal surface, and increased in thickness (4 cms in parts).

Microscopically - Placenta, well marked syphilitic changes; umbilical cord showed round celled infiltration and thickening of media and intima of blood vessels. Chondro epiphysitis in lower end of femur /

femur. Levaditi examination - spirochaetes in liver, spleen and lung.

Cause of death, Syphilis.

CASE 13 :

1-para, aet 32. Admitted to Royal Maternity Hospital on 11/4/26 on account of severe abdominal pain and possible concealed accidental haemorrhage. One hour after admission patient suddenly showed signs of air hunger and general collapse; slight vaginal haemorrhage; uterus boggy and tender - had increased rapidly in size since admission. Abdominal Caesarean section performed, retro-placental haematoma, fibrotic placenta and still-born foetus. History - had excessive sickness during first part of pregnancy; noted oedema of ankles one month before admission, no oedema of face or hands; had frequent frontal headaches (more than when not pregnant); no dimness of vision and no vomiting recently; no urinary symptoms. At 5 a.m. on 11/4/26 she was wakened from sleep by severe lower abdominal pain, which came in spasms; no vaginal haemorrhage, felt faint and cold. Transferred to Royal Maternity Hospital at 10 a.m. Albumen ++ in catheter specimen of urine.

Previous pregnancy normal (twin) children, alive and well.

Premature /

Premature fresh male foetus, weight 1,450 grms, length 38½ cms. Marked congestion of internal organs; subpleural, subepicardial and subcapsular haemorrhages in thymus and liver.

Placenta - marked surface fibrosis; two large interstitial haemorrhages and one large retroplacental haematoma and several small red infarcted areas. Microscopically placenta shewed marked congestion and red infarction. Lungs - small amount of liquor amnii in alveoli; no haemorrhage; no pneumonia.

Cause of death - Asphyxia following placental separation.

Puerperal history. 19/4/26. Blood urea nitrogen and non protein nitrogen - 12 and 27 mg.% Wassermann reaction negative. 28/4/26. Catheter specimen of urine contained trace of albumen. Urea concentration test :

| | | |
|---|---|---|
| (1) | 113 ccs | 1.65 mg |
| (2) | 109 ccs | 1.65 mg. |
| (3) | 56 cms | 2.35 mg. |

CASE 14 :

2-para aet 30. District case. Twin pregnancy. Was quite well during pregnancy until 8/4/26 when she had a slight vaginal haemorrhage and abdominal pain, no clots; sent for district nurse, who said that haemorrhage was only "show". Bleeding continued for 10 days when she went into labour.

Previous /

Previous pregnancies - 1st terminated at 7th month, child still-born; 2nd terminated at 8th month, child still-born.

Wassermann re-action negative; trace of albumen in catheter specimen of urine on 8/4/26 and 20/4/26. 1st child was delivered by the breech and was still-born; 2nd child was vertex delivery and lived 20 hours.

Premature fresh female foetus, weight 1,100 grms, length 37½ cms; lungs not fully expanded; congestion of all internal organs, but no haemorrhage except in right lateral ventricle, where there was a small clot about 1 cm. in size and several small sub-ependymal haemorrhages. There was also a haematoma in left sterno-mastoid muscle. Microscopically - interstitial haemorrhage in lungs. Placenta not obtained.

Cause of death - Cerebral haemorrhage.

Puerperal history. Blood urea nitrogen 150 - 200 mg.% Non-protein nitrogen 250 mg.% Inestimable trace of albumen in catheter specimen. Blood pressure 136/84 mm. Hg. Urea concentration test not obtained.

CASE 15 :

Twin of case 14, lived 20 hours; vertex presentation; spontaneous delivery.

Premature /

Premature fresh female foetus, weight 1,165 grms, length 40 cms. Lungs fully expanded; congestion of all internal organs, but no haemorrhages. Placenta was not obtained.

Microscopically - alveoli and bronchi were filled with liquor amnii, lanugo hairs and epithelial cells; there was also an inflammatory reaction, mainly polymorphonuclear leucocytes and a patchy broncho pneumonia.

Cause of death - pneumonia.

CASE 16 :

Child 10 days old. Was stated to be feeble from birth, and gradually became more so; took breast for 3 days, then had to be put on bottle, as it was apparently unable to suck; two days later pipette feeding was commenced as child was not even able to take bottle. There was no vomiting; bowels were regular; no fits; no cyanosis.

Full-time fresh male foetus, weight 2,745 grms, length 51½ cms. Umbilical cord was separated. Congestion of internal organs; haemorrhage into lower lobe of left lung which was consolidated; subdural clot on under aspect of tempero-spenoidal lobe.

Microscopically - Acute haemorrhagic pneumonia; leucocytic infiltration was present but not to a marked extent.

Cause of death - Acute haemorrhagic pneumonia.

CASE 17 :

District case; 0-para, aet 30.

Premature fresh female foetus, weight 1,995 grms, length 44 cms; no placenta obtained; marked cyanosis; no p.m. rigidity. Lungs were partially expanded; congestion of internal organs, but no haemorrhage.

Microscopically - small amount of liquor amnii and lanugo hairs in lungs; no pneumonia.

Cause of death - Prematurity.

CASE 18 :

0-para, aet 27. Admitted to Royal Maternity Hospital on 15/4/26 for albuminuria of pregnancy. History. Patient was quite well until 8 weeks before admission, when she noted that she had difficulty in getting on her shoes, later she noted that her feet and ankles were swollen. 3 weeks before admission she noted her hands and face were swollen. She had slight frontal headaches, no giddiness, but blurred vision; no urinary symptoms; bowels regular. Was in bed at home for one week before admission. On admission 15/4/26, there was marked oedema of feet and legs, swelling of face, particularly under eyes, and slight oedema on dorsal aspect of hands. No foetal heart heard while patient was in /

in hospital and no "life" felt by mother. Blood pressure 190/120. Catheter specimen of urine contained 1.4% albumen, numerous casts and red blood corpuscles; no bile but acetone ++ in the urine. Blood urea nitrogen 11 mg.%; non-protein nitrogen 23 mg.%. Van den Bergh indirect reaction 0.25 units Phenal-tetrachlor-phthalein test, 4 units at end of 1 hour, 2 units at end of 2 hours after injection. 16/4/26 Blood pressure 180/118; albumen (catheter) 1.2%; acetone ++ . 17/4/26 Blood pressure 172/110; albumen (catheter) 1.4%; acetone ++. Vomited urea given for urea concentration test; went into labour 17/4/26 and was delivered of a premature macerated foetus, weight 2,130 grms; length 44 cms; placenta was small, 195 grms, ratio 10.9. There were numerous small infarcted areas in the placenta, and also a marked surface fibrosis of the maternal surface; thickness of placenta 2 cms. Mother's Wassermann reaction was negative.

Nothing special of note found on post-mortem examination. Liver weighed 70 grms; spleen 3 grms; no chondro-epiphysitis.

Cause of death - Ante-partum asphyxia from placental infarction and ? insufficiency.

Puerperal history of mother - 22/4/26 Albumen .3%; no oedema; Blood pressure 126/80. 23/4/26 Albumen .4%. Reported at hospital 3 months after delivery. There /

There was a trace of albumen in catheter specimen of urine, blood pressure 120/80; Urea concentration test :

| | Total Vol in ccs | mg. urea. |
|---|---|---|
| (1) | 100 | 2.15 |
| (2) | 105 | 2.55 |
| (3) | 63 | 2.2. |

CASE 19 :

O-para, aet 38. Admitted to Royal Maternity Hospital on 21/4/26; Pregnancy normal, vertex presentation; foetal heart heard on admission; foetal heart was not heard one hour before delivery; spontaneous delivery; 1st stage of labour lasted 18 hours; 2nd stage lasted 4½ hours.

Full time fresh female foetus, weight 2,750 grms, length 50½ cms, Marked congestion of all internal organs; numerous petechial haemorrhages, subpleural, subepicardial and diaphragmatic; no dural tear; no cerebral haemorrhage.

Microscopic examination - Lungs, alveoli distended with liquor amnii, epidermal cells, meconium and lanugo hairs; no leucocytic infiltration; no pneumonia, no haemorrhages.

Cause of death - Intra-partum asphyxia.

Puerperal Wassermann reaction negative; blood urea nitrogen and non-protein nitrogen 12 and 26 mg.%. No albumen in urine.

CASE 20 :

O-para, aet 27. Patient stated that she felt quite well during the whole of her pregnancy; she went for a long walk and did her usual household work on day before admission. For 2 or 3 days before admission she had slight frequency of micturition and dysuria, and slight spasmodic pain over left side of lower abdomen; felt "life" last at 10.30 p.m. on 24/4/26. At 11.30 p.m. patient woke up and found that she had had a vaginal haemorrhage; there were no clots of blood, and she had no pain. Vaginal haemorrhage was fairly profuse and continued intermittently until admission to Royal Maternity Hospital at 2.30 a.m. 28/4/26. On admission - edge of placenta felt on vaginal examination; vertex presentation; no foetal heart heard; no albumen in catheter specimen of urine; cervical and vaginal canals packed with gauze; macerated foetus delivered 6 hours later.

Premature ($8\frac{1}{2}$ months) slightly macerated female foetus; marked pallor of whole body; skin peeling off feet and blisters over hands and forehead; all tissues were friable and congested; there was a haemorrhage into medulla of right suprarenal gland, but no haemorrhages elsewhere. Placenta consisted of 3 separate lobes, one large and two smaller ones. The smallest lobe had been a placenta praevia.

Microscopic /

Microscopic examination: Placenta and cord normal; lungs atelectatic; no pneumonia.

Cause of death - Ante-partum asphyxia from placental separation.

Puerperal history: Wassermann reaction negative. Blood urea and non-protein nitrogen 17 and 30 mg.% No albumen in urine.

CASE 21 :

0-para, aet 18. Attended Ante-natal clinic for 4 months before delivery, on account of vaginal discharge. .025% albumen noted in catheter specimen of urine on one occasion only 20.1.26. Blood pressure 110/82. Wassermann reaction negative. Blood urea nitrogen and non-protein nitrogen 18 and 28 mg.%. Admitted to Ante-natal wards on 31/3/26 for version, as breech was presenting, attenpts at version not successful. Re-admitted on 24/4/26 for induction, breech still presenting; bougie induction; 1st stage of labour lasted 18 hours, 2nd stage 2½ hours; foetal heart noted to be irregular at 1 a.m. 25/4/26 and child was extracted; cord was twice round neck.

Full time fresh female foetus, weight 3,720 grms; length 53 cms; marked congestion of all internal organs and /

and numerous petechial haemorrhages, particularly in thorax; thymus was large, weighed 23.5 grms. There was a complete tear of right tentorium cerebelli, and diffuse haemorrhage over lower surfaces of temporal lobes, pons and cerebral peduncles.

Cause of death - Tentorial tear.

CASE 22 :

2-para, aet 31. No antenatal supervision. Admitted to Royal Maternity Hospital at 3 p.m. on 25/4/26, hand presenting shoulder impacted, R.A.A. uterus tender and tending towards tonic contraction. Onset of labour at 7 p.m. 24/4/26; further history indefinite, but was anaesthetized at 9 a.m. 25/4/26 as pain was severe, and no progress was being made. Duration of 1st stage 24 hours, 2nd stage, 45 minutes, 3rd stage 20 minutes. Bipolar podalic version done and foot anchored with clove hitch; spontaneous delivery of body 5 hours later and extraction of shoulders and head. Foetal heart heard on admission and immediately before delivery.

Full time fresh male foetus, weight 3,370 grms, length 51 cms. Congestion of all organs and few petechial haemorrhages in thorax; diffuse blood stained serum over base of brain, but no tentorial tear detected. Microscopically /

Microscopically lungs not expanded; no pneumonia; cord and placenta normal.

Cause of death - Asphyxia probably preceded and was the cause of the cerebral haemorrhage.

Wassermann re-action negative. Blood urea nitrogen and non-protein nitrogen 17 and 18 mg.%.

CASE 23 :

District case, no history obtained.

Full time fresh male foetus, weight 3,545 grms; length 52 cms. Marked lividity and no p.m. rigidity. There was an unusually large caput over upper and posterior part of left parietal bone; congestion of internal organs, but no haemorrhage; there was an incomplete tear of right tentorium cerebelli. Microscopic examination: Alveoli of lungs shewed small amount of liquor amnii and a few lanugo hairs in alveoli; no haemorrhage and no pneumonia.

Cause of death - Tentorial tear.

CASE 24 :

1-para, aet 29. Admitted to Royal Maternity Hospital on 27/4/26, child born as breech as far as shoulders /

shoulders and no pulsation in umbilical cord; cervix fully dilated; head high up; arms were brought down and attempts at traction on head failed; skull perforated and 2 pints of fluid withdrawn and head then delivered.

Full time fresh hydrocephalic foetus, weight 3,520 grms without brain. Marked congestion of internal organs and numerous petechial haemorrhages, subpleural, subepicardial and subcapsular in thymus. There was microscopic evidence of syphilis - placenta, liver, pancreas, umbilical cord and epiphysis. Levaditi examination negative.

Cause of death - intra-partum asphyxia from pressure of uterus and placenta on foetal head. Second cause, probable Syphilis.

Puerperal Wassermann re-action was negative.

CASE 25 :

Baby lived 12 hours; feeble from birth; cyanosed at birth, and required considerable amount of resuscitation; no convulsions.

Premature fresh male foetus, weight 1,710 grms; length 40 cms; talipes equino varus of left foot; lividity and p.m. rigidity; lungs only partially expanded /

expanded; clot 1 x 1½ cms in right lateral ventricle and several petechial subependymal haemorrhages. Congestion of all internal organs; no haemorrhages in lung, but a few subepicardial haemorrhages along subventricular sulcusThere were 2 arteries and 2 veins in umbilical cord. Mother's Wassermann re-action negative. She had no ante-natal supervision; caught cold two days before admission and on admission to hospital temperature 101.6, pulse 127, respirations 38.

Cause of death - Cerebral haemorrhage.

Puerperal Wassermann re-action was negative. No albumen in urine.

CASE 26 :

0-para, aet 25. Attended Ante-natal clinic for last 3 months of pregnancy; Wassermann re-action weak positive on 25/2/26; given .3 N.A.B. on 2/3/26 and Wassermann re-action was negative one week later. Admitted in labour 2/5/26; vertex L.O.A. prolonged labour; given m.iv. pituitrin twice during the 2nd stage, which lasted 6 hours 40 minutes; membranes ruptured 20 hours before delivery; foetal heart was good until just before delivery, when it was noted to be rapid; cord was twice round neck and had to be clamped and cut before shoulders were /

were delivered; baby still-born, with no attempt at respiration. Full-time, fresh, male foetus; weight 2,870 grms; length 53 cms; marked congestion of all internal organs; no subpleural haemorrhages, but a few subepicardial along anterior aspect of intraventricular sulcus; no dural tear and no cerebral haemorrhage.

Microscopic examination: Lungs unexpanded; shew massive catarrhal pneumonia; considerable amount of fibrous catarrhal cells; a few polymorphonuclear and lymphocytic leucocytes; considerable amount of liquor amnii and lanugo hairs in alveoli and bronchioles. Bronchioles also shew a sub-acute inflammatory reaction.

Cause of death - Broncho-pneumonia.

Puerperal Wassermann reaction negative.

CASE 27 :

1-para, aet 39. Admitted to Royal Maternity Hospital on 17/4/26 for albuminuria of pregnancy. Felt well until 2 weeks before admission, when she noted swelling under eyes, then 3 days before admission swelling of feet and ankles; very severe headaches and blurring of vision day before admission. On admission blood pressure 166/100; .44% albumen in catheter specimen of urine; blood urea nitrogen and non-protein nitrogen /

nitrogen 27 and 53 mg.% Urea concentration test

| | | |
|---|---|---|
| (1) | 40 ccs. | .95 mg% |
| (2) | 61 ccs | 1.35 mg% |
| (3) | 32 ccs | 1.95 mg%. |

Kept under observation for 6 days, then labour induced as symptoms increased: 1/5/26 Spontaneous delivery, vertex presentation; baby premature, lived 10 minutes.

Premature fresh male foetus, weight 700 grms, length 36 cms; marked lividity and p.m. rigidity; Placenta was firm, avascular and had numerous small white infarcts; thickness between 1 and 2 cms; cotyledons not well marked. Microscopically: marked endarteritis obliterans in blood vessels and old degeneration and necrosis of villi in parts; marked congestion of all internal organs; lungs were partially expanded, and microscopically there were interstitial haemorrhages into lung substance. There was a small clot and subependymal haemorrhage in right lateral ventricle.

Cause of death - Cerebral haemorrhage.

Puerperal history: On day following delivery the oedema had practically disappeared; amount of urine passed had considerably increased; blood-pressure 180/110 mm.Hg; 15/5/26 blood-pressure 160/100 mm.Hg. albumen .05% in catheter specimen. (vomited urea for test)

CASE /

CASE 28 :

Baby lived 3 days. District case, no history obtained. Premature fresh male foetus, weight 1020 grms, length 28 cms; marked lividity and p.m.rigidity. There was no naked eye evidence of congestion; there was a small amount of free fluid in pericardial cavity, but no subepicardial haemorrhage; there was a small patch of consolidation at upper part of lower lobe of left lung. Microscopically - patchy catarrhal pneumonia.

Cause of death - Catarrhal pneumonia.

CASE 29 :

1-para, aet 21. Admitted to Royal Maternity Hospital in labour; no antenatal supervision; vertex presentation, spontaneous delivery; baby lived 20 mins.

Premature fresh male foetus; weight 850 grms; length 36 cms; post-mortem rigidity and lividity; placenta appeared normal; no infarction; no haemorrhage; no fibrosis. Microscopically: normal appearance, no endarteritis obliterans, no enlargement of villi and no fibrosis; marked congestion of all internal organs and numerous subepicardial haemorrhages; large subcapsular haemorrhage in right suprarenal gland.

Cause of death - Prematurity.

Mother's /

Mother's Wassermann reaction negative. No albumen in urine; blood urea nitrogen and non-protein nitrogen 9 and 15 mg.%; urea concentration test normal; average quantity = 75 ccs; average m.g.% urea = 2.3. Previous pregnancy had terminated at 7th month; child lived 1½ hours; no history of toxaemia.

CASE 30 :

District case - no name and no history.

Premature fresh male foetus; no p.m. rigidity; very large caput succedaneum over right parietal; weight 1,085 grms; length 37½ cms; marked congestion of internal organs and subpleural petechial haemorrhages; lungs not expanded; haemorrhage into middle third of falx cerebri; both tentoria cerebelli intact; no haemorrhage into ventricles. Microscopic examination: Lungs - a few of the alveoli are distended with liquor amnii, squamous cells and lanugo hairs; no pneumonia, but numerous small interstitial haemorrhages.

Cause of death - Intrapartum asphyxia.

CASE 31 :

1-para, aet 27. District case. Previous pregnancy ended prematurely; no history of toxaemic symptoms; baby /

baby died aet 4 days. Present pregnancy (twins). Was quite well until 3 weeks before delivery, when she noted that she had difficulty in opening her eyes in the morning, and that her face was swollen; about the same time she noted that her hands and ankles were swollen; during last week of pregnancy patient stated that she had frequency of micturition, dysuria, and passed a scanty amount of urine; during last 2 days she had severe frontal headache and saw specs in front of eyes. Went into labour on 10/5/26, and was delivered spontaneously. Child 1., still-born (R.S.A.); (Child 2 alive (L.S.A)) Premature female foetus, macerated, probably dead for some days; weight 875 grms, length 36 cms. The tissues were soft and organs difficult to dissect. There was a complete tear of both tentoria cerebelli; brain tissue and membranes were friable: Placenta was firm and avascular, particularly over area belonging to macerated foetus, no haemorrhage, but infarctions. Microscopically: one section showed normal placenta, and another two sections shewed marked endarteritis obliterans of blood vessels and definite early red infarction; marked distension of the villi and a few red corpuscles and homogenous mass in intervillous spaces.

Cause of death - Ante-partum asphyxia from placental infarction.

Puerperal /

Puerperal history. 11/5/26 Considerable amount of oedema of feet and ankles, slight oedema of face, distinct puffiness under eyelids; blood urea nitrogen 10 mg.% non-protein nitrogen 24 mg.% .8% albumen in catheter specimen 25/5/26. Urea concentration test (1) 2 mg.urea %; (2) 2.4 mg.urea %; (3) 2.5 mg.urea % volume not measured as total quantity not sent.

CASE 32 :

0-para, aet 29. Admitted to Royal Maternity Hospital on 11/5/26 in labour; no ante-natal supervision. L.O.A. Labour commenced at 10 p.m. 11/5/26, membranes ruptured at 11 p.m 12/5/26; spontaneous delivery of still-born foetus at 10.50 a.m. 13/5/26. Full time fresh female foetus, weight 2,855 grms, length 52 cms; marked lividity and no p.m. rigidity. Well marked congestion and numerous haemorrhages, subcapsular and interstitial in thymus; subpleural and interstitial in lungs (both naked eye and microscopically); numerous sub-epicardial haemorrhages, most marked at apex of heart and around great vessels; haemorrhages along posterior thoracic wall and superior surface of diaphragm. The only haemorrhage found in the abdomen was on the superior aspect of the bladder. Brain /

Brain. Well marked congestion of all cerebral vessels, but no evidence of haemorrhage. There was a small incomplete tear of right tentorium cerebelli.

Cause of death - Intra-partum asphyxia.

2nd cause -Tentorial tear.

CASE 33 :

0-para, aet 22. No ante-natal supervision. Admitted to Royal Maternity Hospital in labour on 11/5/26. R.O.A. 1st stage lasted 23¾ hours; 2nd stage 7 hours; 3rd stage 20 minutes; membranes ruptured 20 hours before delivery; low forceps applied on account of failing foetal heart; still-born child delivered; full time fresh, male foetus; weight 3,430 grms; length 54 cms; marked lividity; no p.m. rigidity; congestion of internal organs but no evidence of haemorrhage; lungs were macerated, lower part of trachea and both bronchi were filled with meconium. Abdomen: The bladder extended up to the level of the xiphisternum, very thinned wall; no stricture of the urethra; no phimosis; no hydronephrosis. No cerebral lesion. Microscopically - Lungs: Alveoli and bronchi filled with liquor amnii, lanugo hairs and meconium; numerous epidermal cells in bronchi. There are several areas with polymorphonuclear leucocytic /

leucocytic infiltration, but no pneumonia.

Cause of death - Asphyxia from aspirated liquor amnii.

Puerperal Wassermann reaction negative. Blood urea nitrogen and non-protein nitrogen 17 and 30 mg.%.

CASE 34 :

6-para, aet 38. first 4 pregnancies normal, full time spontaneous deliveries. 5th pregnancy complicated with albuminuria, treated in Royal Maternity Hospital, and pregnancy terminated at 8th month; child lived 3 weeks. 6th pregnancy also complicated with albuminuria, but treated during the whole of pregnancy and symptoms were not severe, but oedema of hands, face and feet were present. 6th pregnancy terminated October 20th 1921. Present pregnancy: Patient states that from the 4th month she had oedema of hands and face (patient was quite definite about the occurrence of oedema at this early date) About the 4th month she noticed that urine was highly coloured and scanty; from 5th month she has had severe frontal headaches. She had no headaches when not pregnant. Patient has dieted since the 4th month, and since end of 5th month has been under medical supervision. Previous to admission /

admission patient was confined to bed for 3 weeks on account of oedema and headaches. She has always been constipated; teeth are in a very bad condition. Admitted to Royal Maternity Hospital on 28/7/26; premature labour complicated by transverse presentation and albuminuria; blood pressure 182/162; 1.2% albumen in catheter specimen of urine, specific gravity 1012; 30 ozs passed in 24 hours. External version was attempted but failed - membranes ruptured and internal version done, transverse converted into cephalic presentation, cord prolapsed into vagina while this was being done, and it was found to be pulseless; spontaneous delivery half an hour later. Premature fresh female foetus, weight 1,350 grms, length 42 cms; marked lividity; no p.m. rigidity: Placenta: About three-quarters of placenta was ploughed up by haemorrhage into it, mainly interstitial; there were two old yellow infarcts and several small recent infarcts: Signs of asphyxia with numerous haemorrhages - subpleural, subepicardial, subcapsular into thymus and subperitoneal between layers of broad ligament.

Cause of death - Intra-partum asphyxia from prolapsed cord from malpresentation.

29/7/26 Blood urea nitrogen and non-protein nitrogen 15 and 30 mg.% Wassermann reaction negative. 34 ozs urine passed. 7/8/26. Urea concentration test

(1) /

| | | |
|---|---|---|
| (1) | 95 ccs | 2.5 mg.% (urea) |
| (2) | 46 ccs | 2.65 mg.%(urea) |
| (5) | 67 ccs | 2.45 mg.%(urea) |

Trace of albumen in catheter specimen of urine.

CASE 35 :

2-para, aet 30. Sent for examination from Deaconess Hospital. Wassermann reaction negative; no albuminuria; blood urea nitrogen = 15 mg.%. Blood non-protein nitrogen = 30 mg.% Mother has felt perfectly well during pregnancy; no hydramnios; face presentation; membranes ruptured 10 hours before delivery; child still-born; anencephalic fresh female foetus, weight 2,150 grms; length 45 cms; marked lividity of face and neck; no spinal defect; palate was highly arched, and cleft to 2nd degree; there was a rudimentary cerebrum which weighed 2.5 grms, but no cerebellum was distinguished naked eye. Thyroid weighed 1.5 grms and was congested; thymus weighed 16 gms, shewed subcapsular haemorrhage; lungs were ~~macer~~ una-erated and deeply congested; numerous subpleural haemorrhages; the suprarenals were rudimentary - the right weighed .5 gms, the left .75 gms; large amount of subcutaneous fat was noted throughout. Microscopically: Lungs shewed very little liquor amnii and lanugo hairs in alveoli, but there was a well marked pneumonia; alveoli filled with few /

few polymorphs, large number of mononuclears, red blood cells and fibrin.

Cause of death - pneumonia.

CASE 36 :

1-para, aet 23. Last pregnancy terminated at the 5th month, one month after a threatened miscarriage. No antenatal supervision during this pregnancy; has felt well; no history of syphilis obtained; spontaneous delivery of a premature foetus, who died 36 hours later.

Premature fresh female foetus; weight 2,050 grms; length 38 cms; marked lividity and p.m.rigidity. The superficial layers of the skin of the feet and of the hands were almost entirely separated off the deeper layers, in some parts there were large blisters,(about the size of a threepenny piece) into which haemorrhages had occurred. The legs and arms were covered with a coppery polymorphous rash. On the left forearm there was a definitely indurated area and the centre of this nodule had broken down. Thorax: Free fluid in pleural and pericardial cavities; marked congestion of all organs; lungs partially aerated; pneumonia alba in both lungs. Heart: Few petechial subepicardial haemorrhages /

haemorrhages along interventricular sulcus. Abdomen: Liver firm in consistence; congested on section; doubtful fibrosis round periportal tracts; weight of liver 80 grms: Spleen: weight 15 grms, enlarged and congested; definite perisplenitis on its lower border; Pancreas was enlarged, pale in colour and very firm in consistence; doubtful chondro epiphysitis. Brain: Haemorrhage into middle third of falx cerebri. Microscopic examination: Placenta:- definite syphilitic changes: Pancreas:- slight increase in interstitial fibrous tissue; islets of Langerhans not increased in number: Kidneys and suprarenals: no evidence of specific infection; no haemorrhage. Thyroid: extensive proliferation of interacinar stroma, complete destruction of accini in parts, in other parts a few acini are seen, but contain little or no colloid; vacuolated cells fill the lumen of several of the acini. There is a slight lymphocytic infiltration. Blood vessels shew proliferation of media, but no round celled infiltration. Thymus: the majority of Hassal's corpuscles appear normal; a few contain polymorphonuclear leuco cytes and others are replaced entirely by granular debris. There is no increase of stroma cells. Spleen: edge of thickened capsule seen in section shews marked inflammatory changes; hyperemia of spleen pulp.

Liver: /

Liver: well marked periportal cirrhosis with lymphocytic infiltration and also a less well-defined cirrhosis. Lungs: partially aerated; definite pneumonia alba. Levaditi examination positive.

Cause of death - Syphilis.

CASE 37 :

Baby lived 8 days. Child 'failed' from birth, lost weight daily; did not vomit; bowels were not regular; was jaundiced from 3rd day; no increase in respirations noted.

Mother was admitted to Hospital on account of revealed and concealed accidental haemorrhage. History was that she had been well except for pain over upper part of uterus for 3 weeks previous to admission. On 8/5/26 she had severe vaginal haemorrhages at 11 a.m. and 2 p.m. On admission to Hospital slight vaginal haemorrhage still going on; pain over fundus; trace of albumen in catheter specimen of urine; blood pressure 146/96. R.O.A. delivered spontaneously 6 hours after admission. Wassermann negative. Premature jaundiced male foetus, weight 1,460 grms, length 44 cms. Naked eye examination revealed some consolidation of lungs; no cerebral haemorrhage. Microscopic examination of lungs /

lungs revealed broncho-pneumonia. Placenta weighed 345 grms, shewed no evidence of infarction, but there was a large retroplacental haematoma.

Cause of death - Broncho pneumonia.

Puerperal history: No albumen in urine on 11, 17 and 19/5/26. Blood urea nitrogen and non-protein nitrogen, 10 and 17 mg %. Vomited urea given for test.

CASE 38 :

4-para, aet 37. Admitted to Royal Maternity Hospital for concealed and revealed accidental haemorrhage and lateral placenta praevia. First 3 pregnancies went to full time; 1st and 3rd children died from birth injuries; 2nd was still-born; 4th pregnancy terminated in a miscarriage at 3 months. Present pregnancy: Came to antenatal clinic on one occasion 29/3/26, when she stated she had no symptoms, albumen negative, Wassermann reaction negative; blood pressure 124/80. On 13/5/26 she thought foetal movements were not so active, also she noted that her hands were swollen and limbs 'felt stiff' and eyelids were difficult to open in the morning. There was no history of urinary symptoms nor headache nor swelling of ankles. On 17/5/26 she did not feel well all /

all day - no definite symptoms, but thought she had caught cold, and had headache. On 18/5/26 she woke up at 6 a.m. and on attempting to micturate passed two large clots of blood per vaginam, no pain associated. Seen by doctor at 6.30 a.m., who said pulse was quite good and advised patient to stay in bed. At 7 a.m. labour pains began; at 7.30 felt ill, vomited and had severe vaginal haemorrhage; bled intermittently until admitted to Royal Maternity Hospital. On admission 1.2% albumen in catheter specimen of urine; blood pressure 146/80; no foetal heart heard; teeth carious; not constipated. 10 a.m. Caesarean section performed. Premature macerated male foetus was delivered, weight 2,240 grms; length 47 cms. The lower part of the placenta overlay the lower uterine segment; placenta shewed numerous infarcts; in the region of the left cornu evidence of thrombosis was seen and the adjacent broad ligament was markedly oedematous.

Cause of death - Ante-partum asphyxia from placental infarction.

Puerperal history of mother: 20/5/26 Blood urea nitrogen 17 mg.%. Non-protein nitrogen 29 mg.% 24/5/26 Blood pressure 146/80. Albumen ++ in catheter specimen. 30/5/26 Albumen + in catheter specimen. Blood pressure 110/82. 31/5/26 Urea concentration test

| | Total vol ccs | Mg.urea %. |
|---|---|---|
| (1) | 50 | 1.5 |
| (2) | 110 | 1.5 |
| (3) | 50 | 1.8 |

9/6/26 day of discharge from hospital. albumen+ in catheter specimen; blood pressure 120/84. Reported 11/8/26. No albumen in urine; blood pressure 128/90. Urea concentration test

| | Total vol.in ccs | Mg.urea % |
|---|---|---|
| (1) | 180 | 1.2 |
| (2) | 85 | 1.75 |
| (3) | 80 | 2.15. |

CASE 39 :

0-para, aet 37. Admitted to Royal Maternity Hospital in labour on 19/5/26. L.O.A. .5% albumen in catheter specimen, blood pressure 124/90; no evidence of toxaemia; D.C. 4"; os 5 fingers dilated; membranes ruptured, no history of time of rupture obtained; head overlapping brim; put on twilight sleep. 11.30 a.m. 20/5/26, no advance, contour of abdominal wall shewed that uterus was contracted down on child; foetal heart good, rate 130; liquor amnii meconium stained. Classical Caesarean section performed; foetus delivered with difficulty owing to contraction ring round the neck of the foetus; difficulty not due to fixation in the brim, as head not reached on vaginal examination; extension of uterine incision through the contraction ring allowed of delivery of the foetus. Fresh post-mature male foetus; well developed centre of ossification in cuboid; no p.m. rigidity /

rigidity; well marked lividity. Thorax: marked congestion of all internal organs and subpleural haemorrhages round root of lungs and subepicardial haemorrhages along interventricular sulcus. Lungs unaerated. Abdomen: nothing special to note; Liver ratio 20.2; Spleen ratio 289. Brain: no haemorrhages and no dural tear; Spinal cord: Cruciate ligament round 2nd cervical vertebrae was incompletely torn from its attachment on the right side, and there was haemorrhage along anterior surface of spinal cord as far as 7th cervical vertebrae. Microscopic examination: Umbilical cord shewed proliferation of intima and media and marked lymphocytic infiltration of both arteries and veins. Placenta shewed uniform increase in size of villi, with diminution of intervillous spaces, and endarteritis changes in main stem vessels. Lungs shewed no evidence of syphilitic change. Epiphysis was not examined microscopically.

Cause of death - Tear of cruciate ligament.

(2) Probable syphilis.

CASE 40 :

Twin of case 21, lived 7 days. Premature fresh female foetus, weight 900 grms, length 37 cms. Umbilical cord completely separated; marked lividity and p.m. rigidity. /

rigidity: Thorax: free fluid in pleural cavity; lungs fully expanded, felt solid, particularly posteriorly and markedly haemorrhagic looking, particularly in the lower lobes posteriorly. Heart: Free straw coloured fluid in pericardial cavity; subepicardial haemorrhages into tricuspid valve only. Brain: Some congestion of cerebral vessels, but no haemorrhages. Microscopic examination: Lungs shewed acute haemorrhagic pneumonia, very extensive, covering practically the whole of the lung; polymorphonuclear leucocytes were infiltrated throughout the lung: Liver shewed either cloudy swelling or advanced p.m. changes.

Cause of death - Acute haemorrhagic pneumonia.

CASE 41 :

4-para, aet 27. History of 4 previous normal pregnancies, spontaneous deliveries, children alive and well. Present pregnancy: Sickness up to the 6th month, otherwise pregnancy has had a normal course. At 12 noon on 22/5/26 she had a sudden severe vaginal haemorrhage, followed shortly after by abdominal pain, which soon passed into labour pains. Transferred to Royal Maternity Hospital at 2 p.m., still oozing freely from vagina: .2% albumen in catheter specimen of urine, blood pressure 140/100. Vagina packed;

Classical /

Classical Caesarean section was performed at 6 p.m. as packing was unsuccessful and uterine tone was lost. No foetal heart heard. Macerated foetus delivered; placenta completely separated at time of operation. Full time macerated foetus, weight 2,540 grms, length 47 cms. Maceration was not of lomg standing, skin was peeling off feet only. Thorax: Free blood-stained fluid in pleural cavity; very numerous subpleural haemorrhage; lungs unaerated; several small subcapsular haemorrhages into thymus. No haemorrhage in abdominal cavity. Brain: Excess of cerebro-spinal fluid over surface of brain, but no congestion of cerebral vessels. Placenta: about three-quarters was infarcted - several old and a few recently infarcted areas. Microscopic examination: Infarction and marked congestion of inter-villous spaces; endarteritis obliterans of blood vessels.

Cause of death - Ante-partum asphyxia from placental infarction and separation.

Puerperal history of mother. Mother was too ill on admission for blood test and she died on 3/6/26. No investigations were carried out.

CASE 42 :

9-para, aet 38. 8 normal pregnancies, spontaneous deliveries and normal puerperia. 6th pregnancy normal /

normal, but labour lengthy and instrumental delivery. Present pregnancy normal throughout, showed no signs of toxaemia; no oedema, no headaches; no urinary symptoms; for 2 weeks before admission she had slight pain over lower abdomen. At 10.30 a.m. on 22/5/26 pain became severe, and she passed several bloodclots from vagina. Sent to Royal Maternity Hospital. On admission no haemorrhage occurring; not in labour; foetal heart not heard; .2% albumen in catheter specimen of urine, blood pressure 126/90. Mother felt no foetal movements after haemorrhage at 10.30 a.m. 23/5/26. L.O.A. spontaneous delivery, almost precipitate. Full time macerated male foetus, weight 2,650 grms, length 48 cms; marked lividity; no p.m. rigidity. Placenta 475 grms, covered practically entirely with large retro-placental haematoma; placental surface infiltrated with blood clot; no infarction; no interstitial haemorrhage. Thorax: free blood stained fluid in pleural and pericardial cavities; numerous subpleural, subepicardial and subcapsular haemorrhage into thymus. Abdomen: Subcapsular haemorrhage of liver and medulla haemorrhage of right suprarenal. Brain: Marked congestion of all vessels and haemorrhage over left cerebral hemisphere towards base of brain and left tempero-spenoidal region.

Cause of death - Ante-partum asphyxia from placental separation by haemorrhage.

Puerperal /

Puerperal history. No investigations were carried out. 30/5/26 blood pressure 126/90; albumen + in catheter specimen. 3/6/26 albumen + in catheter specimen. Patient was written to but did not report.

CASE 43 :

0-para, aet 27. History of several attacks of rheumatic fever. Present pregnancy: marked morning sickness during first 3 months of pregnancy, then quite well until middle of April, when she noted that she became breathless on slight exertion, flushed readily, and was easily tired. On 15/5/26 she was admitted to Royal Infirmary with an attack of heart failure; for 3 weeks before admission she had increasing breathlessness on exertion and sleeplessness; oedema of feet and legs for 2 months before admission to R.I.E. On 22/5/26 admitted to Royal Maternity Hospital, face flushed and cyanosed; marked breathlessness; pulse 100 - 120 irregular in time and force; oedema of feet legs and abdominal wall; dilation of veins on chest wall; dilatation of left side of heart; mitral pre-systolic and diastolic murmurs; no foetal heart heard; mother had felt no foetal movements for one week. Labour induced 25/5/26, membranes ruptured and hand prolapsed into vagina; bipolar podalic version done, spontaneous delivery /.

delivery. Premature macerated female foetus, weight 1030 grms; length 41 cms; free blood stained fluid in pleural, pericardial and peritoneal cavities; congestion of organs; few haemorrhages, subpleural, subepicardial and subcapsular in thymus and in left suprarenal. Brain was very friable and could not be examined minutely. Both tentorium cerebelli were completely torn. Placenta: showed no infarcts, looked healthy both on naked eye and microscopic examination, except for rather more surface fibrosis than usual.

Cause of death - Ante-partum asphyxia following maternal cardiac failure.

CASE 44 :

0-para, aet 17. No antenatal supervision. Admitted to Royal Maternity Hospital on 26/5/26 in labour; transverse presentation; hand prolapsed into vagina; foetal heart heard on admission. After great difficulty internal version was done and child extracted. Foetal heart failed during manipulations. Full time fresh male foetus, weight 2,920 grms, length 54 cms; marked lividity and no p.m. rigidity. There was congestion, but no haemorrhage in internal organs. Brain: Left tentorium cerebelli was completely torn and /

and covered with blood clot; right tentorium cerebelli was incompletely torn; haemorrhage into middle third of falx cerebri, close to its junction with tentorium cerebelli. Microscopically: Lungs not expanded; small amount of liquor amnii; no pneumonia. Placenta: endarteritis obliterans, but villi normal; some hyaline degeneration near termination of villi.

Cause of death - Tentorial tear and haemorrhage.

Puerperal history: Wassermann reaction negative. Blood urea nitrogen and non-protein nitrogen 9 and 19 mg.%

CASE 45 :

0-para, aet 20. No antenatal supervision. Admitted in labour on 28/5/26; twin pregnancy with hydramnios. Wassermann re-action negative. No albumen. Blood pressure 125/84; no toxaemic signs or symptoms. L.O.A. spontaneous delivery. Premature macerated female foetus, weight 910 grms; length 37 cms; well marked lividity and p.m. rigidity. The maceration was well advanced, and made dissection difficult; free blood-stained fluid in pleural, pericardial and peritoneal cavities; no evidence of haemorrhage in lungs and heart; skull was markedly misformed, probably due to pressure during delivery; it was impossible to examine brain, as tissues tore during dissection.

Placenta /

Placenta: twin uniovular. There were several interstitial haemorrhages, and one infarcted area over part of the placenta, which nourished the macerated foetus; each of these features were seen on microscopic examination also.

Cause of death - Ante-partum asphyxia from placental infarction.

Puerperal history of mother. 30/5/26, Blood urea nitrogen 11 mg.% Non-protein nitrogen 21 mg.% Wassermann reaction negative. No albumen in urine. 9/6/26. Urea concentration test :

| | Total vol. ccs. | Mg.urea % |
|---|---|---|
| (1) | 85 | 2.0 |
| (2) | 100 | 2.5 |
| (3) | 65 | 2.5 |

CASE 46 :

5-para, aet 38. Five normal pregnancies and spontaneous deliveries. Present pregnancy normal until 22/5/26 when she had a slight vaginal haemorrhage, but no pain, then slight intermittent haemorrhage for a week; on 27/5/26 she had a severe vaginal haemorrhage at 6.30 p.m., labour pains started shortly afterwards. Vaginal haemorrhage continued, though slight, up to time of admission. 27/5/26. On admission, oozing a little from vagina, bled freely on examination, placenta completely covered os, which was 2 fingers dilated.

Foetal /

Foetal heart 150, feeble and irregular. No albumen. External version done, placenta gone through and foot brought down; foetal heart after manipulations 100. Spontaneous delivery 3½ hours later. Slightly premature fresh male foetus, weight 2,445 grms; length 49 cms; signs of asphyxia were well marked; petechial haemorrhages - subpleural, subepicardial and subcapsular in thymus. No dural tear; blood stained serum in left temporo-spenoidal region and extending towards base of brain. Microscopically: Lungs were not expanded; no liquor in alveoli; no haemorrhage; no pneumonia. There was only one artery and one vein in umbilical cord. This condition was unfortunately not discovered until microscopic examination of cord was being done.

Cause of death - Intrapartum asphyxia from placenta praevia.

CASE 47 :

Twin of case 45. Lived 5 hours; L.O.A. spontaneous delivery, precipitate. Premature fresh female foetus, weight 1,140; length 39 cms; marked lividity and p.m. rigidity. There was a large caput and marked overlapping of cranial bones. Thorax: Lungs partially expanded; no haemorrhage; free fluid in pericardial cavity; few petechial subepicardial haemorrhages along interventricular sulcus and along great /

great blood vessels. Abdomen: congestion of organs; no haemorrhage. Brain: There was a tear of falx cerebri in its middle third, extending into sagittal suture; haemorrhage extended down both sides of the tear, but most marked on left side; there was a complete tear of left tentorium cerebelli, and a bruising of right tentorium cerebelli; there was haemorrhage on tentoria, must marked on left side and extending down in front of the cerebellum. Microscopically: Lungs not fully expanded; alveoli and bronchi contained a little liquor amnii and lanugo hairs.

Cause of death - Tentorial tear and cerebral haemorrhage.

CASE 48 :

3-para, aet 30. 3 previous pregnancies uncomplicated and labours normal. Present pregnancy: Excessive vomiting during whole of pregnancy; oedema of right leg for 2 months; no difficulty with micturition; no headache. Labour pains began at 4 p.m. 27/5/26; membranes ruptured at 11 p.m. on 27/5/26; admitted to R.M.H. on 29/5/26, face presenting, no foetal heart heard; spontaneous delivery at 6 p.m. 29/5/26 of an anencephalic foetus. Anencephalic macerated postmature male foetus, weight 3,240 grms, length 44 cms; centre /

centre of ossification in cuboid was well defined; large amount of subcutaneous fat; maceration was of some days standing. The usual 9 features of anencephalic syndrome were present. There was in addition well defined signs of asphyxia in thorax; subpleural, subepicardial, and haemorrhages along posterior thoracic wall. Thymus gland weighed 19.5 gms. Decomposition in abdomen made structures difficult to define, but there was no evidence of haemorrhage apparent. Suprarenals were present, but both were rudimentary. No evidence of brain tissue could be obtained.

Cause of death - Anencephaly.

Puerperal history. Wassermann reaction negative. Blood urea nitrogen and non-protein nitrogen 13 and 23 mg.% No albumen in urine.

CASE 49 :

2-para, aet 34. 2 previous pregnancies and labours normal. Present pregnancy was normal until 24/5/26 when ? membranes ruptured. Clear fluid continued to come away intermittently until night of 38th, when patient woke up to find that she had a vaginal haemorrhage. There was no pain. Own doctor called in who stated that os was closed and also packed vagina and /

and transferred patient to Royal Maternity Hospital. 31/5/26. On admission vaginal pack saturated and slight oozing; no foetal heart heard. .8% albumen in catheter specimen; blood pressure 180/155. Put on quinine grs x. two hourly. Spontaneous delivery of a macerated foetus 6 hours later. Premature macerated male foetus, weight 1,610 grms, length 46 cms; maceration not well advanced; foetus probably dead for 12 hours; marked lividity; no p.m. rigidity. Thorax: tissues were infiltrated and oedematous; Thymus was small, weighed 1.5 grms; Pleural and pericardial cavities contained blood-stained serum; few subpleural and subepicardial haemorrhages. Abdomen: maceration well advanced, liver very friable. Brain: soft and oedematous, dura tore readily. Both tentoria were torn. Placenta contained only a small area of normal placental tissue. About three-quarters of the total placental surface was firm and avascular, and in addition there were several old infarcts. No haemorrhages. Microscopically placenta showed localized necrosis, marked congestion in intervillous spaces, endarteritis obliterans well defined; chorion infiltrated with leucocytes, mainly polymorphs.

Cause of death - Ante-partum asphyxia from placental infarction.

Puerperal /

Puerperal history of mother: 2/6/26 Blood urea nitrogen = 15 mg.%; non-protein nitrogen = 25 mg.% Wassermann reaction negative; blood pressure 184/156. Albumen + in catheter specimen of urine. 6/6/26 Blood pressure 170/136. Albumen still present in urine. Vomited urea given for test on two occasions. Reported 11/8/26, blood pressure 128/90. Urea concentration test

| | Total Vol.ccs | Mg.urea % |
|---|---|---|
| (1) | 105 | 1.0 |
| (2) | 85 | 1.5 |
| (3) | 70 | 1.25. |

CASE 50 :

0-para, aet 22. No antenatal supervision. Admitted to Royal Maternity Hospital in labour; impacted breech; no albumen in catheter specimen. Labour commenced at 11.50 p.m. 30/5/26; breech extracted at 10 p.m. 31/5/26. Premature (7½ months) fresh female foetus, weight 1040 grms; length 38½ cms. Marked lividity and no p.m. rigidity. Thorax: no free fluid in pleural cavity; no haemorrhage in thoracic organs, except interstitial haemorrhage in lung; small amount of free straw coloured fluid in pericardium; no subepicardial haemorrhage. Abdomen: marked congestion, but no haemorrhage. Brain: There was a small clot about 1 cm. in size over lower part of right cerebral hemisphere; dura was intact; no /

no haemorrhage into ventricle. Placenta shewed marked infarction and interstitial haemorrhages. Microscopically: Lungs shewed massive interstitial haemorrhages; no liquor amnii in alveoli; no pneumonia.

Cause of death - Cerebral haemorrhage.

Puerperal history: Wassermann reaction negative. Blood urea nitrogen and non-protein nitrogen 11 and 18 mg.% No albuminuria. Vomited urea given for test.

CASE 51 :

0-para, aet 28. Patient had suffered from deafness from age of 5 years, attributed to an accident at that time. Her eyesight was not good, but she said that she had never had eye trouble. Wassermann reaction during pregnancy and after delivery negative. Her intelligence was markedly subnormal.
Present pregnancy: Attended antenatal clinic on one occasion, 17/5/26 when her pregnancy was judged to be normal. Foetal heart heard; no albumen; Blood pressure 120/86. Admitted in labour 3.40 p.m. 7/6/26. Labour had commenced at 10 p.m. 6/6/26. On admission liquor amnii was stained and offensive. Twins delivered at 5.p.m. 7/6/26, both breech presentations. No albumen in urine; blood pressure 130/90.

Premature /

Premature (7½ months) macerated male foetus, weight 1,100 grms; length about 38 cms: maceration was very advanced; skin of scalp and the dural membranes had broken down during delivery and brain tissue had escaped: tissues were very soft and infiltrated with blood-stained serum: free blood-stained serum in all cavities: liver was shrunken and brownish grey in colour; weight ratio 44; spleen ratio 733.3. Placenta weight 380 grms: there was extensive fibrosis of the maternal surface; cotyledons were not well marked; thickened from maternal to foetal surface; one cord had a central insertion and the other a velamentous insertion. Microscopically: Umbilical cord shewed thickening of intima and media of vein and round celled infiltration of vein and arteries. Placenta shewed definite syphilitic changes, enlargement of terminal villi, diminished intervillous spaces and blood supply. Epiphyses shewed deepening of zone 3 and irregular calcification. Levaditi examination negative.

Cause of death - probable syphilis.

Puerperal history: 8/6/26 Blood urea nitrogen 12 mg% Non protein nitrogen 28 mg.% No albumen in urine. Blood pressure taken on two occasions 126/84.

18/6/26. Urea concentration test

| | Total vol. in ccs. | Mg.urea % |
|---|---|---|
| (1) | 100 | 1.4 |
| (2) | 105 | .95 |
| (3) | 105 | 1.00. |

CASE 52 :

Twin of case 51. Premature macerated male foetus, weight 1,700 grms, length 40 cms. Maceration seemed almost more advanced than in case 51; abdominal wall almost black in colour. Brain was so soft and friable that it could not be examined. Liver ratio 35.2. Spleen ratio 800. Microscopic examination: Umbilical cord shews lymphocytic infiltration of vein and arteries: Placenta see case 51: Epiphyses, not so marked as in case 51, but deepening of zone 3 is present.

Cause of death - probable syphilis.

CASE 53 :

3-para, aet 28. 1st pregnancy full-time instrumental delivery; 2nd pregnancy full-time, complicated labour, cause not known; 3rd pregnancy full-time, spontaneous delivery. Present pregnancy: no antenatal supervision; onset of labour at 4 a.m. 5/6/26, membranes ruptured at 9.45 a.m., and cord prolapsed into vagina; transferred to Royal Maternity Hospital. Admitted at 10.15 a.m. Os three-quarters dilated, cord presenting at vulva; practically pulseless, ceased pulsation during examination; head well down in pelvic cavity and firmly fixed /

fixed. Spontaneous delivery of a still-born foetus half an hour later. Full-time fresh female foetus, weight 3,750 grms, length 55 cms; marked lividity, but no p.m. rigidity. Placenta: Cotyledons not specially well defined; no evidence of fibrosis of placenta; no infarction; no haemorrhage. Very well marked signs of asphyxia, numerous petechial haemorrhages in lung, heart, thymus and liver. Both ureters and pelvis of kidneys were dilated; - minor degree of hydronephrosis. Microscopic examination: Lungs - alveoli and bronchi were filled with liquor amnii, lanugo hairs and epidermal cells: there was no pneumonia, but in a few areas there was a definite polymorphonuclear leucocytic infiltration. Placenta and cord normal.

Cause of death - Asphyxia following prolapse of cord.

Wassermann reaction negative. Blood urea nitrogen and non-protein nitrogen 14 and 37 mg.%

CASE 54 :

District case, no history obtained. Child had lived 36 hours. Premature (8½ months) fresh female foetus, weight 2,140 grms, length 46 cms, marked lividity, especially round head and neck. p. m. rigidity. Thorax /

Thorax: Thymus weighed 13 grms, congested, a few petechial haemorrhages on posterior surface. Lungs: no free fluid in pleural cavity, lungs partially aerated, markedly congested, but no consolidation. Heart: Free fluid in pericardium; marked haemorrhages towards base of heart; foramen ovale was patent, and there was a small clot in right auricle near foramen ovale. Abdomen: Liver markedly congested; 2 small subscapular haemorrhages; Brain: marked congestion of cerebral vessels; one small subependymal haemorrhage in right ventricle. Microscopic examination - Lung showed a small amount of liquor amnii in alveoli; massive pneumonia with marked polymorph. and mono nuclear leucocytic infiltration.

Cause of death - Pneumonia.

A feature of this case was the generalized enlargement of the lymphoid tissue throughout the body. The tonsils were the largest seen in this series, and also the mesenteric glands were the largest seen; the thymus gland was not enlarged.

CASE 55 :

O-para, aet 25. No ante-natal supertision. R.O.A. position; low forceps,(easy delivery) on account of /

of maternal distress. Full time fresh male foetus, weight 3,900 grms; length 56 cms; marked moulding in occipito-frontal direction of head. A considerable time was taken during the delivery of the head, and the foetal heart was observed frequently and was 140 - 160, never below 110. The heart was beating when child was born, but no attempt at respiration was made. Congestion in all organs, but no haemorrhages. Brain: Complete tear of left tentorium cerebelli, and incomplete tear of right tentorium cerebelli. Marked congestion of cerebral vessels but no evidence of haemorrhage. Microscopic examination of lungs shewed very little liquor amnii in bronchi; lungs completely unexpanded. Placenta was normal both on naked eye and microscopic examination.

Cause of death - tentorial tear.

Wassermann reaction negative. Blood urea nitrogen and non-protein nitrogen 14 and 29 mg.%. Urea concentration test :

| | | |
|---|---|---|
| 1. | 30 ccs | 3.05 mg.% urea |
| 2. | 90 ccs | 3.25 mg.% urea |
| 3. | 40 ccs | 3.00 mg.% urea |

CASE 56 :

7-para, aet 41. District case. Previous pregnancies /

pregnancies normal, all children alive and well. Present pregnancy: Never felt well during pregnancy, no vomiting, but feeling of malaise and tiredness; no toxaemia symptoms; no headaches, no urinary symptoms etc. No albumen in urine, blood pressure 142/118. Did not feel life during last 5 or 6 weeks of pregnancy. Full time macerated male foetus, weight 3,690 grms, length 52½ cms. Maceration was well advanced, and tissues were very soft and oedematous. Liver weighed 110 grms; spleen weighed 4 grms; no epiphysitis. Placenta shewed no evidence of syphilis either on naked eye or microscopic examination; no haemorrhage and no infarcts, but fibrosis of maternal surface of the placenta was present. Microscopic examination - marked endarteritis obliterans and hyaline degeneration, most marked in chorion at terminal villi.

Cause of death - Ante-partum asphyxia.

Wassermann reaction negative. Blood urea nitrogen and non-protein nitrogen 12 and 24 mg.%.

CASE 57 :

6-para, aet 33. Previous diseases - treated in Stobhill Sanatorium for 3 months, treated for ? tubercular knee and ? tubercular ulceration of face

4 /

4 years ago. Main points in history - Good health up to 6 years ago, always a little nervous, married 15 years ago; 6 years ago had a still-born child, since then reluctant to meet people; treated for gumma of thyroid, and signs of toxic thyroid two years ago. Admitted to Pilton Hospital 8/2/26 for intensive treatment of syphilis. Wassermann reaction +++ strongly positive; treated as an out-patient from 3/4/26 to 11/5/26. On 6/6/26 she complained of headache and was jaundiced; on 16/6/26 became restless, almost maniacal, transferred to Ward 3 R.I.E., became unconscious about 3 p.m. 11/5/26 and was transferred to antenatal ward, Royal Maternity Hospital. On admission: In comatose condition; marked jaundice skin and conjunctivae; eyeball protruding; mouth open and tongue hanging out; petechial haemorrhage into skin; teeth carious and in a very bad condition; liver dullness diminished; knee jerks absent, Babinski positive on right side; catheter specimen of urine albumen + bile++, granular casts, pus cells and bacilli; no acetone, no sugar; in labour, os 3 fingers dilated; delivered spontaneously, breech, macerated foetus. Patient died at 10.45 p.m., of acute pulmonary oedema. Premature macerated male foetus, weight 810 grms, length 35 cms; maceration was advanced, foetus must have /

have been dead for several days. Tissues were infiltrated with blood stained serum, there were a few small haemorrhages in lungs but not in any other organs. Placenta was not greasy; cotyledons were not well marked, placenta was thickened from maternal to foetal surface. Microscopic examination: placenta shows areas of typical syphilitic infection, but this was not uniform: umbilical cord - there was a lymphocytic infiltration of vein only, arteries were not thickened and appeared normal. Definite chondro-epiphysitis.

Cause of death - Syphilis.

CASE 58 :

2-para, aet 22. 1st pregnancy normal; 2nd pregnancy terminated at 6th month. Present pregnancy: marked sickness for first 3 months; on 11/6/26 was knocked down by motor and right ~~tibula~~ tibia and fibula fractured; transferred to Royal Maternity Hospital same day in labour. On admission: Slight vaginal haemorrhage; uterus tense and tender; trace of albumen in catheter specimen; blood pressure 140/95; L.O.A. Spontaneous delivery of a still-born foetus; birth of placenta was preceded by expulsion of 2 large clots; no retro-placental clot; placenta expelled immediately after clots /

clots, premature (8 months) fresh male foetus, weight 2,400 grms, length 49 cms, marked lividity, but no p.m. rigidity. Thorax: Thymus congested, and had numerous subcapsular haemorrhages, it extended further up into the neck on the right side than normally, and had a bulbous termination at bifurcation of common carotid artery: Lungs were ~~ma~~ unaerated and had numerous petechial subpleural haemorrhages: Heart: free straw-coloured fluid in pericardial cavity, subepicardial haemorrhage. Abdomen: Congestion of all organs; horse-shoe shaped kidney. Brain: Dura intact, but blood-stained serum over base of brain. Microscopic examination: lungs not expanded; little liquor amnii in alveoli; no pneumonia. Placenta was normal on both microscopic and naked eye examination.

Cause of death - Asphyxia due to placental separation at time of accident.

Patient was transferred to Royal Infirmary after delivery and no investigations were carried out.

CASE 59 :

1-para, aet 30. Previous pregnancy normal. Came to Ante-natal Clinic on one occasion 12/1/26; pelvic measurements normal, no albumen in urine, blood pressure 126/86; Wassermann reaction negative. Had been /

been quite well during pregnancy; no toxaemic symptoms, and no history of syphilis; admitted on 12/3/26 in labour; L.O.A. position, spontaneous delivery, full-time macerated female foetus, weight 4,010 grms, length 52 cms; tissues were infiltrated with blood-stained serum, free blood-stained serum in all cavities; organs congested, but shewed no haemorrhage; ratio of liver and spleen 33.4 and 364 respectively; no chondro-epiphysitis. Placenta was not obtained, weight at hospital 736 grms, ratio 5.4. Umbilical cord shewed no lymphocytic infiltration on microscopic examination.

Cause of death - Ante-partum asphyxia.

CASE 60 :

0-para, aet 20. No ante-natal supervision; admitted to Royal Maternity Hospital baby born as far as umbilicus, head and posterior arm extracted after admission. Full-time fresh female foetus, weight 3,000 grms, length 52 cms; marked lividity. There was marked congestion of all internal organs and numerous petechial haemorrhages - subpleural, subepicardial and subcapsular in Thymus. Brain shewed marked congestion in all vessels and small incomplete tear in right tentorium cerebelli. No evidence of epiphysitis. Microscopic examination: Placenta and cord normal. Lungs: alveoli distended /

distended with liquor amnii, lanugo hairs and epithelial cells. No evidence of pneumonia and no leucocytic infiltation present.

Cause of death - Intra-partum asphyxia from pressure of uterus and placenta on after-coming head.

CASE 61 :

7-para, aet 36. First 4 pregnancies normal full-time, spontaneous deliveries, children alive and well. 5th pregnancy ended at 8th month, child lived 24 hours; 6th pregnancy terminated at 4th month; 7th pregnancy went to full-time, child alive. Was in Royal Maternity Hospital for 7th pregnancy - Wassermann reaction was weak positive before delivery: placenta weight ratio was 2., puerperal Wassermann reaction negative. Present pregnancy: No ante-natal supervision, delivered on district of a macerated foetus. Never felt well during this pregnancy, no oedema of ankles, but general malaise. Says she never felt "life". Premature macerated ($7\frac{1}{2}$ months) female foetus, weight 1,700 grms, length 45 cms. Maceration was very far advanced, skin was greyish brown in colour, lips nearly green. All organs were pale in colour, soft and very friable. Brain could not be removed as a whole. Definite chondro epiphysitis. Placenta /

Placenta: weight 300 grms, ratio 5.6. Cotyledons were well marked; there was a considerable amount of fibrosis of maternal surface; placental area was thickened from maternal to foetal surface. Microscopic examination: Chondro-epiphysitis: Placenta: enlargement of chorionic villi, endarteritis obliterans, diminution of intervillous spaces, hyaline degeneration and small celled infiltration of chorion. Umbilical cord: No lymphocytic infiltration of blood vessels; walls of blood vessels shew degenerative changes. Levaditi examination of organs spirochaetes .

Cause of death - Syphilis.

Puerperal Wassermann reaction strongly positive

CASE 62 :

Sent from Cowgate Dispensary - no particulars. Premature ($7\frac{1}{2}$ - 8 months) fresh male foetus, with generalized oedema, weight 2,545 grms, length 43 cms. The oedema was most marked at ears, back of hands, dorsal aspect of feet and over abdominal wall; tongue protruded from mouth. Thorax: Considerable amount of free fluid in subcutaneous tissues, free fluid in pleural cavity, lungs macerated; right side of heart dilated; vessels congested and several petechial haemorrhages. Thymus /

Thymus small, only 1.5 grms and congested. Abdomen: Free straw-coloured fluid in peritoneal cavity; some congestion of internal organs, but no haemorrhages. Liver was firm, reddish-brown in colour, slightly enlarged, weighed 90 grms, ratio 28.2. Spleen was firm, weighed 9.5 grms, ratio 267.8. Brain: There was marked oedema of scalp, but no excess of fluid in ventricles. No epiphysitis either on naked eye or microscopic examination. Placenta: weight 415 grms, ratio 6.1; the surface was smooth, there were no infarcts and no haemorrhages; cotyledons were well marked, edge of the placenta was thickened from maternal to foetal surface. Microscopic examination showed normal structure. Umbilical cord was oedematous. On microscopic examination the oedema was well marked and extended into the walls of the blood vessels.

Cause of death - General foetal oedema.

Maternal Wassermann reaction was negative. The pregnancy was said to have been normal; delivery was spontaneous.

CASE 63.

6-para, aet 40. 1st to 3rd pregnancies normal. 4th and 6th pregnancies complicated with albuminuria, both /

both children died aged 24 hours. 5th pregnancy terminated at 3rd month. Previous health: Operation for gall stones in 1921, had trouble with kidneys for last 5 years. No supervision during present pregnancy, and patient states that she had oedema of legs since 4th month of pregnancy; she had had difficulty with micturition for some time, she did not know how long; just before admission to hospital she had frequent headaches, and her sight was not so good as it used to be. Seen by her doctor for the first time on 24/6/26 and transferred to Royal Maternity Hospital same day. On admission: well marked oedema of legs, ankles and abdominal wall, .8% albumen in catheter specimen of urine, blood pressure 146/92; specific gravity of urine 1015. Went into labour same night and delivered spontaneous (1) vertex died 1 day later (2) breech, still born. Baby lived 18 hours, marked cyanosis from birth. Full time fresh male foetus, weight 2,650 grms, length 51 cms, marked lividity and p.m.rigidity. Thorax: Lungs were fully expanded, very congested and there seemed to be small consolidation posteriorly; congestion of blood vessels round heart, but no haemorrhage. Abdomen: Congestion of organs, no haemorrhage; Brain: No dural tear and no cerebral haemorrhage. Microscopic examination: Lungs were fully expanded and contained /

contained a considerable amount of liquor amnii, epidermal cells and lanugo hairs. There was patchy pneumonia. Placenta not obtained.

Cause of death - Pneumonia.

Puerperal history. 25/4/26. Blood urea nitrogen and non-protein nitrogen 10 and 24 mg.% 28/4/26 ·1% albumen in catheter specimen Blood pressure 130/80 mm.Hg. 28/7/26 No albumen, Blood pressure 124/80 mm.Hg. Urea concentration test :

| | | | |
|---|---|---|---|
| (1) | 85 | ccs. | 1.9 mg.% urea |
| (2) | 106 | ccs | 2.45 do. |
| (3) | 66 | ccs. | 2.2 do. |

CASE 64 :

1-para, aet 27. 1st pregnancy terminated at 2nd month. No supervision during present pregnancy; sent into hospital from district on account of considerable vaginal haemorrhage; no history of primary sore; no symptoms of specific disease. Baby lived 2 days, was feeble from birth. Premature (7½ months) fresh jaundiced male foetus, 740 grms, length 26 cms. Marked lividity and p.m. rigidity. There was little made out on post-mortem examination, except doubtful epiphysitis. Brain: No haemorrhage or dural tear. Microscopic examination - Chondro epiphysitis. Liver: periportal cirrhosis /

cirrhosis and lymphocytic infiltration: Lungs: Thickening of alveolar wall; marked fibrosis and total destruction of alveoli in parts; lymphocytic infiltration in parts. Pancreas: increase in fibrous tissue and slight lymphocytosis. Thyroid: few normal acini seen; marked proliferation of fibrous tissue into interacinar spaces; the lumen of acini are filled with cubical cells. Spleen: hyperaemic; little change in Hassal's corpuscles. Kidneys: marked congestion in medulla. Umbilical cord shewed lymphocytic infiltration of media of vein and arteries. Placenta: uniform enlargement of villi; diminution of intervillous spaces and diminished blood supply.

Cause of death - Probable Syphilis.

Wassermann reaction taken 3 weeks after delivery, strongly positive.

CASE 65 :

2-pare, aet 28. 1st and 2nd pregnancies normal, last pregnancy August 1923. Present pregnancy: Slight amount of vomiting in first 3 months. Came to antenatal clinic on 18/6/26 complaining of pain in left side, heartburn and frequency of micturition; no headache, no oedema, blood pressure 198/120; trace of albumen /

albumen in catheter specimen of urine. L.O.A. position; foetal heart heard to left of umbilicus. Although advised to stay in Hospital patient would not do so. Wassermann reaction negative. Returned to clinic 1/7/26, blood pressure 182/120, trace of albumen and sugar in catheter specimen of urine; no foetal heart heard. 7/7/26 delivered on district of a macerated premature ($7\frac{1}{2}$ - 8 months) male foetus, weight 1,230 grms, length 43 cms. maceration was well advanced; liver ratio 61.5, spleen ratio 820; Placenta 220 grms, ratio 5.5, small, firm and avascular; cotyledons not well marked; numerous recent and old infarcts. Microscopic examination of placenta revealed same conditions as were seen naked eye. Umbilical cord: fibrosis of arteries and vein; arteries were practically occluded; no lymphocytic infiltration of vessel wall; no chondro epiphysitis.

Cause of death - Antepartum asphyxia from placental infarction.

Reported 13/8/26. Blood pressure 148/90. No albumen. Wassermann reaction again taken - result negative. Would not wait for urea concentration test.

CASE 66 :

Twin to Case 63. Full time fresh female foetus, weight 3,350 grms, length 51 cms., marked lividity, no p.m. rigidity /

rigidity. Thorax and abdomen: well marked congestion of all organs, and a small amount of free fluid in pleural, pericardial and peritoneal cavities; there was no haemorrhage. Brain: No dural tear or cerebral haemorrhage seen. Microscopic examination: Lungs, alveoli were distended, but did not contain liquor amnii.

Cause of death - Intrapartum asphyxia.

CASE 67 :

12-para, aet 38. 1st pregnancy, full time spontaneous delivery, child lived one hour. 2nd pregnancy terminated at 7th month, child still born. 3rd pregnancy full time child, died aged 3 weeks. 4th, 5th and 6th pregnancies full time spontaneous deliveries, children alive, 7th and 8th pregnancies full time children, still born; 9th pregnancy terminated at 8th month, child died aged one month. No history of previous illnesses; no history of syphilis obtained: Present pregnancy: no morning sickness during first 3 months, but brown vaginal discharge which cleared up spontaneously at end of 3rd month. Felt quite well until one week before admission - felt 'out of sorts' headache, but no other signs or symptoms of toxaemia. Admitted to Royal Maternity Hospital on 30/7/26. Vertex presentation, foetal heart /

heart heard. No albumen in urine, specific gravity of urine 1012, blood pressure 120/70. Teeth were carious, marked pyorrhoea alveolaris. Spontaneous delivery of a full time fresh hydrocephalic male foetus with spina bifida in lumbar region, weight 2,400 grms without brain, length approximately 50 cms, marked lividity and no p.m. rigidity. The hydrocephalous was of a slight degree - anterior fontanelle was enlarged, cranial bones separated, deficient ossification in cranial bones, marked distension of both lateral ventricles and of 4th ventricle, the cerebral tissue over lateral ventricle was thinned out to thickness of parchment paper; no dural tear, no cerebral haemorrhage. There was a meningocele from 1st to 3rd cervical vertebrae, and a myelomeningocele from 1st to 5th lumbar vertebrae. The lower extremities shewed no obvious bony deformity, but shewed marked lividity. Thorax: Marked signs of asphyxia and numerous petechial haemorrhages. Abdomen: Slight enlargement of spleen and liver. Horse-shoe shaped kidney. No naked eye evidence of epiphysitis. Microscopic examination revealed secondary evidence of syphilis, and spirochaetes were found on Levaditi examination. Placenta - microscopic examination negative.

Cause of death - Syphilis.

CASE /

CASE 68 :

District case, child born before arrival of nurses. Premature fresh female foetus, weight 2,390 grms, length 49 cms, marked lividity, no p.m. rigidity. Marked signs of asphyxia - subpleural, subepicardial and subcapsular haemorrhage in thymus. Lungs fully expanded. Placenta: weight 860 grms, ratio 2.6, cotyledons not well marked, not fleshy looking, thickened from foetal to maternal surface, no infarctions. Microscopic examination - no evidence of syphilis. Lungs: alveoli dilated and filled with liquor amnii, epidermal cells and lanugo hairs; slight inflammatory reaction in one part. Wassermann reaction taken 3 weeks after delivery - negative.

Cause of death - Intrapartum asphyxia.

CASE 69 :

6-para, aet 31. Came to Antenatal Clinic on 16/3/26 complaining of a yellow vaginal discharge (pus cells +++, gram positive diplococci and streptococci) Attended Clinic regularly with no signs of toxaemia until 12/7/26, when she noted that legs were swollen. No albumen in urine, blood pressure 130/90, the highest during her pregnancy. Admitted. Membranes ruptured at

at 12.40 a.m. 13/7/26, pains very slight; os fully dilated at 10 a.m. Low forceps applied at 12 mid-day owing to irregularity in foetal heart rate and non-advance of head. Baby very cyanosed when born, and did not respond to treatment. Full time fresh male foetus, weight 3,740 grms, length 54 cms. There was marked congestion of all organs, but no haemorrhages. Brain: There was a complete tear over right tentorum cerebelli, and clot overlying cerebellum. Microscopic examination. Alveoli were distended with liquor amnii, epidermal cells and lanugo hairs: no inflammatory reaction: no pneumonia and no haemorrhage.

Cause of death - Tentorial tear and haemorrhage.

CASE 70 :

3-para, aet 35. 1st pregnancy 1913, full time spontaneous delivery, child died $\frac{10}{12}$ yrs. cause meningitis. 2nd pregnancy 1920, full time spontaneous delivery, child alive and well. 3rd pregnancy 1922 terminated in a miscarriage at 3rd month. History: Patient had a primary sore in February 1921. She treated it with Eusol; six weeks after she had a generalized rash. Doctor diagnosed syphilis, and gave her treatment, which consisted of 3 injections and mercury pills for one /

one year. Patient was then told she was cured. Present pregnancy: Patient had a slight amount of vomiting for first 3 months, she felt 'life' in March. Came to Ante-natal Clinic on 27/4/26 because she had not felt 'life' for a fortnight, and she thought she was getting smaller. Uterus was at the level of the umbilicus, no foetal heart was heard, no albumen in urine, blood pressure 120/96, Wassermann reaction strongly positive. Was treated for syphilis at Clinic until 19/7/26, when she went into labour spontaneously. No foetal heart was heard, and uterus decreased slightly in size. 19/7/26, Spontaneous delivery of a premature macerated male foetus, weight 570 grms, length 34½ cms. Placenta: weight 130 grms, cotyledons well marked, surface not fleshy looking, not thickened, thickened and firm in consistence and a considerable amount of surface fibrosis. No epiphysitis on naked eye examination. Microscopic examination: Placenta - most typical section seen. Umbilical cord - marked fibrosis of blood vessels, no lymphocytic infiltration: Epiphysitis - marked degree. Levaditi examination of organs, spirochaetes not found.

Cause of death - Probable syphilis.